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UNDULANT FEVER IN AUSTRALIA.

By W. J. PENFOLD, M.B., Ch.M. (Edinburgh), D.P.H., B.Hyg. (Durham), M.R.C.S. (England), L.R.C.P. (London),

AND

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(From the Baker Institute of Medical Research, Alfred Hospital, Melbourne.)

WE have examined without success THE MEDICAL JOURNAL OF AUSTRALIA from its inception to find references to Malta or undulant fever occurring in Australia. Contagious abortion of bovines is, however, very prevalent and Albiston[1] has shown that infection of the market milk of Melbourne with *Brucella abortus* is widespread. From this milk he has isolated, through the guinea-pig, many strains of *Brucella abortus*.

The close relation of *Brucella melitensis* and *Brucella abortus* has been pointed out by Evans[2] and it is now well known that both organisms can cause undulant fever.

We desire to bring two cases to the notice of the Australian medical profession.

On November 18, 1929, F.B., a man, thirty years of age, was admitted to the Alfred Hospital under the care of Dr. Henry Laurie by whose kind permission we are publishing the notes of the case.

Case I.

Personal History.

For three weeks immediately before admission the patient had suffered from rigors, sweats and fever. He was born in Queensland, but when his present illness commenced nearly two years ago, he was resident in South Australia.

At eight years of age he left Queensland, but not before he had suffered from an attack of nephritis with œdema. During his life there he was in contact with pet goats, but apparently has had no contact with goats since. He suffered as a child from measles and mumps.

In 1918, while in a military camp near Adelaide, he suffered from scarlet fever for which he was isolated for six weeks. At this camp he was vaccinated.

He has spent his life since leaving Queensland in South Australia and in Victoria. Between 1918 and 1928 he enjoyed excellent health and during the period was able to play "League" football. From 1916 to 1928 he was engaged as a salesman and later as departmental manager in a store in Adelaide; his business did not take him into contact with animals or animal products. He has never suffered from venereal disease.

History of Present Illness.

At the end of 1927 the patient suffered from many worries of a business and personal nature. In January, 1928, he began to suffer from shivers, sweats, high temperature, headache and constipation. The fever lasted for eleven weeks, during which time he was confined to bed in Brighton, South Australia. His private physician was of opinion that he was suffering from enteric fever of an atypical character. The agglutination test was not carried out owing to a mishap to the container in transit. After he had been confined to bed for two and a half weeks, a consultant was called in who diagnosed diaphragmatic pleurisy. After leaving his bed, it was two months more before he could walk properly and even then he had not recovered his normal health and strength. He then went for holidays to Peterborough, South Australia, and as he was still not well, to Katoomba, New South Wales, for three weeks. On returning through Sydney he fell ill again and required to go to bed for a few days. It was believed in Sydney that he was suffering from influenza. He ultimately got back to Adelaide on June 28, in time to report to his place of business on June 30.

Until September, 1928, he continued in indifferent health, when he was required to come to Melbourne on private business. While here, he again fell ill with high fever and headache, faintness, thirst, constipation, shivering and sweating and his medical attendant diagnosed the condition as gastric influenza. In November, 1928, he returned to Adelaide. His health remained indifferent and in April, 1929, he again took seriously ill and required to go to bed for one week. During this time he suffered from similar symptoms to those he had had previously and when he got up he did not recover well.

A few months later he took up a position in Melbourne as a wood-turner, but had to give it up after a very short period because of further illness, the chief symptoms of which were palpitation and dropping of heart beats, pains in the stomach and a great feeling of weakness and exhaustion. His medical attendant diagnosed ulcer of the stomach.

In October, 1929, he went to a dairy farm in Gippsland for a holiday. On his fourth day there he had another feverish attack with severe headache, dry mouth and throat, feelings of faintness and malaise. It was necessary for him to go to bed and his temperature on one occasion reached 40·5° C. (105° F.). His medical attendant diagnosed the condition as septic throat. He remained in bed until he was admitted on November 18 to The Alfred Hospital. While on the dairy farm in Gippsland, he drank large quantities of raw milk, but it seems unlikely that this was responsible for his infection. One is more inclined to the view that the illness started at the beginning of 1928 and continued intermittently until the present time and that it was undulant fever.

On admission to The Alfred Hospital his temperature was found to be 39·1° C. (102·4° F.), his pulse rate was 96 and his respirations numbered 29. He appeared pale, dull and apathetic and his face was bathed in perspiration. His pupils were normal, his tongue coated in the centre with a white, thick fur. Examination of his chest revealed no serious abnormality of the lungs or heart. No rose-coloured spots could be seen on abdominal examination. The liver and spleen were not palpable nor were they enlarged as far as could be ascertained by percussion. Examination of the nervous system revealed nothing of importance, except the mental torpor before mentioned.

The X ray examination of the abdomen revealed nothing abnormal. During the time the patient was in hospital he did not show very marked fever. Nevertheless, a study of Chart I will show periods of fairly constant fever alternating with periods of less constant temperature. For example, from November 19 to 26 the diurnal excursion was about 0·5° C.. From November 27, on the other hand, until December 10, a period of about a fortnight, the diurnal fluctuation was more nearly 1° C., to be followed again by a period of relatively constant temperature from December 12 to 20, after which time the differentiation between periods of varying temperature could not be made out so well.

Laboratory Examination.

A blood count soon after admission revealed 5,260,000 red blood corpuscles per cubic millimetre. The hæmoglobin was slightly lower than normal and the white cells numbered 5,600 per cubic millimetre. A differential count by Dr. John McLean, Giemsa's method, revealed:

Neutrophile polymorphs	51·5%
Eosinophile polymorphs	1·5%
Lymphocytes	30·0%
Atypical lymphocytes	1·0%
Monocytes	13·5%
Metamyelocytes	2·5%

The examination of blood smears revealed no malarial parasites. Attempts at ordinary blood culture in broth and bile media resulted in negative findings. On MacConkey's medium no pathogenic organisms were obtained from the fæces nor yet from the urine. The

Wassermann test yielded no reaction. Agglutination tests of the patient's serum against emulsions of *Bacillus typhosus*, *Bacillus paratyphosus A* and *B* and *Bacillus proteus* X19, in dilutions from one in ten to one in 640, all gave no results. Agglutination with *Brucella abortus* emulsion, however, yielded a strongly positive result in serum dilutions from one in 80 to one in 2,560. An emulsion of *Brucella melitensis* also reacted in serum dilutions of one in 80 to one in 1,280. Three normal sera in dilutions of one in 40 to one in 640 gave no reaction when tested against these same emulsions. These results, suggesting undulant fever, were reported to the ward on the fourth day after the admission of the patient.

On December 11, 1929, four attempts at blood culture were made as follows in Huddleston's liver media:

Blood Culture A: Five cubic centimetres of blood were added to one hundred cubic centimetres of Huddleston's liver broth which was incubated aerobically. No growth occurred in this culture during three weeks. After this time a subculture was attempted from it on Huddleston's liver agar, but it remained sterile.

Blood Culture B: Half a cubic centimetre of fresh blood before clotting was spread over the surface of a liver agar plate (Huddleston) and incubated aerobically. This plate showed no growth during three weeks.

Blood Culture C: Five cubic centimetres of blood were added to one hundred cubic centimetres of Huddleston's liver broth and incubated in an atmosphere containing 5% to 10% of carbon dioxide. This remained sterile during three weeks of observation and its attempted subculture at this period was likewise fruitless.

Blood Culture D: As in the case of blood culture B, 0·5 cubic centimetre of blood was spread over the surface of one of Huddleston's liver agar plates and incubated in an atmosphere containing from 5% to 10% of carbon dioxide. This plate showed signs of growth after seven days. The colonies were few, round and opaque and were found to consist of Gram-negative cocco-bacilli, morphologically resembling *Brucella melitensis*. Subcultures were made on to Huddleston's liver agar. Some of these were grown aerobically and some in an atmosphere of carbon dioxide as before. The subcultures grown aerobically did not develop, but those grown in 5% to 10% carbon dioxide grew well.

From a liver agar culture the following set of carbohydrate media was inoculated: Isodulcite, arabinose, dextrose, lævulose, galactose, mannose, maltose, lactose, saccharose, raffinose, inulin, amygdalin, glycerin, erythrite, adonite, dulcite, mannite, sorbite. Not one of these carbohydrates was fermented though the organism grew well.

In milk the organism developed an alkaline reaction. On liver agar no pigmentation of the colony was seen, though control cultures of *Brucella melitensis* caused typical pigmentation.

Absorption tests of patient's serum showed that a known *Brucella abortus* removed the agglutinins both for itself and *Brucella melitensis* more efficiently than did a known *Brucella melitensis* strain, but neither by absorbing patient's serum with *Brucella abortus* nor *Brucella melitensis* were we able to distinguish definitely and certainly the type of organism that we had recovered from the patient.

By December 24 the organism that we had isolated from the liver agar plate, grew quite well in aerobic subculture. This comparatively sudden adaptation to aerobic growth appears to be unusual in the case of *abortus* strains generally. It is supposed to occur usually only after a few months.

The strain we had obtained from the patient was then tested for agglutination against dilutions of the patient's serum over a range from one in 40 to one in 2,560. No reaction occurred. The strain was thereafter subcultured twice a week on Huddleston's liver agar and was found to be still inagglutinable on January 6, 1930, but by January 28 it was agglutinated "+++" by the patient's serum in a dilution of one in 640 and "+" in a dilution of one in 1,280. This agglutination was identical whether it was performed with patient's fresh serum or with patient's carbolized preserved serum which had been taken previously on November 27. The evidence, therefore, suggested that during this time the inagglutinable organism had become completely agglutinable.

On December 28 a second blood culture was taken, the same technique as had been used on December 11 being used. The broth culture and agar plate which were incubated in 5% to 10% carbon dioxide, each showed on January 6 evidence of the growth of a Gram-negative cocco-bacillus. These strains were subcultured in an atmosphere of carbon dioxide and on January 30 the two strains were tested with patient's serum when they were both agglutinated in a dilution of one in 1,280, so that apparently the patient harboured at various times agglutinable and inagglutinable strains of the organism. This new strain was tested on the same sugars as before and was shown to be of the alkaligenes type and produced alkali in milk.

At no time have any of these strains shown any brown coloration of the colonies on liver agar or serum agar medium. No organisms were obtained from the blood cultures that were incubated without carbon dioxide, while of those incubated with carbon dioxide three gave us positive results. Two *melitensis* strains and three *abortus* strains were available for testing against the patient's serum, but direct agglutination tests by this method did not enable us to determine as to whether *Brucella abortus* or *Brucella melitensis* was the infecting agent. On that account, therefore, *melitensis* and *abortus* sera were prepared in rabbits and absorption tests done according to the technique described by Ross in the *Journal of Hygiene*,[2] with the results set out in the accompanying table.

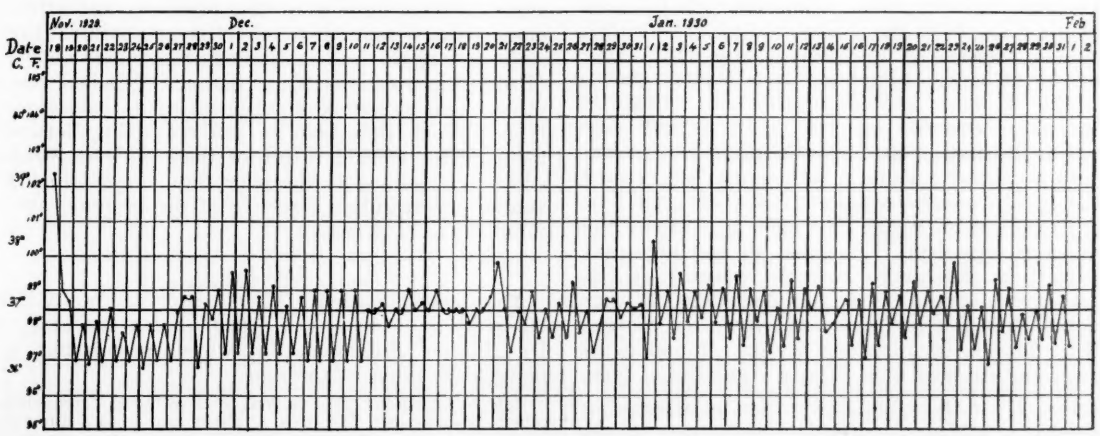

CHART I.

TABLE SHOWING ABSORPTION TESTS BY ROSS'S TECHNIQUE TO DETERMINE THE TYPE OF THE AUSTRALIAN STRAIN F.B.

Section I.

Abortus serum was prepared in an English rabbit by means of *Brucella abortus* (G.P.) and showed a "+++" titre of one in 1,280. The agglutination reactions were carried out with one, two, three and four times the titre strength of serum.

Organism.	Unabsorbed Serum Simple Agglutination.				Serum Absorbed by Test Organisms and Tested against the Homologous Organism.				Serum Absorbed by Various Test Organisms and Tested against the same Organisms to Demonstrate that the Absorption had Actually Occurred.			
	4T.	3T.	2T.	1T.	4T.	3T.	2T.	1T.	4T.	3T.	2T.	1T.
Br. abortus (G.P.)	+++	+++	+++	+++	–	–	–	–	–	–	–	–
Br. abortus (1690)	+++	+++	+++	+++	–	–	–	–	–	–	–	–
Br. abortus (B.B.)	+++	++	+	+	+++	+++	+++	+++	+	tr.	tr.	tr.
F.B.	+++	+++	+++	+++	–	–	–	–	–	–	–	–
Br. melitensis (Sydney)	+++	+++	+++	+	+++	+++	++	+	–	–	–	–

Section II.

Melitensis serum was prepared in an English rabbit against *Brucella melitensis* (V.S.). Titre "+++", one in 1,280.

Br. abortus (G.P.)	+++	+++	+++	+++	+++	+	–	–	–	–	–	–
Br. abortus (1690)	+++	+++	+++	+++	+++	+	–	–	–	–	–	–
Br. abortus (B.B.)	+++	+++	++	tr.	+++	+++	+++	+	–	–	–	–
F.B.	+++	+++	+++	+++	+++	+	–	–	–	–	–	–
Br. melitensis (V.S.)	+++	+++	+++	+++	–	–	–	–	–	–	–	–
Br. melitensis (Sydney)	+++	+++	+++	+++	–	–	–	–	–	–	–	–

Section III.

F.B. serum was prepared in an English rabbit against our strain isolated by blood culture. Titre "+++", one in 2,560.

Br. abortus (1690)	+++	+++	+++	+	–	–	–	–	–	–	–	–
Br. abortus (B.B.)	++	++	+	–	+++	+	tr.	–	tr.	tr.	–	–
F.B.	+++	+++	+++	+++	–	–	–	–	–	–	–	–
Br. melitensis (V.S.)	+++	+++	+	–	++	+	–	–	–	–	–	–
Br. melitensis (Sydney)	+++	+++	+	–	++	+	–	–	–	–	–	–

NOTE.—Saline and serum one in 25 dilutions were used in the case of all emulsions to show that none of them agglutinated spontaneously.

Section I of this table shows that all our *Brucella abortus* and *Brucella melitensis* strains and our own F.B. are agglutinated by the serum made in the rabbit through the injection of *Brucella abortus* (G.P.) and that this serum when absorbed by these various strains loses the corresponding agglutinins, though in the case of *Brucella abortus* (B.B.) the loss is by no means complete. It is further shown that the *abortus* serum absorbed by *Brucella melitensis* or *Brucella abortus* (B.B.) is still capable of agglutinating the *Brucella abortus* organism used to prepare it, while if this serum is absorbed by the known *abortus* strains (G.P.) or (1690) or by our own strain, it can no longer agglutinate the homologous organism. From this we conclude that our strain is similar to *Brucella abortus* (G.P.) and *Brucella abortus* (1690) and definitely different from the *Brucella melitensis* and *Brucella abortus* (B.B.). This latter organism we have found to be atypical and we look upon it as a transitional type.

Section II of the table shows that by absorption tests with a known monotypical *melitensis* serum our strain behaves similarly to the *abortus* strains. In this section it is seen again that *Brucella abortus* (B.B.) has not absorbed much of the *melitensis* agglutinin.

Section III of the table shows that our strain, when injected into a rabbit, produces agglutinins more closely related to *abortus* agglutinins than *melitensis* agglutinins. Here again *Brucella abortus* (B.B.) behaves in a curious manner. We look upon this strain, as before mentioned, as a transitional type and of little help in differentiating between the strains. Consideration of these various results makes us inclined to the view that our strain F.B. is almost certainly a *Brucella* of the *abortus* rather than of the *melitensis* type.

Case II.

The second patient, W.O., entered The Alfred Hospital on February 7, 1930, under the care of Dr. Walter Summons, by whose permission we publish the notes of the case.

Before admission the patient had suffered from three febrile attacks, the first two of about one week's duration each, the third of fourteen days' duration. Between each of these attacks he was apparently well for about one week.

During the third attack of fever his admission to the hospital occurred.

The following were points of interest in his remote history. He had never resided outside of Australia except for a period of twelve months spent in the Solomon Islands fourteen years ago. During his stay in the islands he had contracted malaria. As a child he had suffered from pleurisy. Two years before admission he had an attack of fish poisoning and he has been subject to frequent attacks of "gastric influenza." By trade he is a railway porter and has had no further contact with milk or animals than this particular trade would occasion.

On admission, he was found to have a rapid pulse and respiration, slight elevation of temperature and a furred tongue, but no obvious serious physical signs. The spleen was not enlarged and no tubercle bacilli were detected in the sputum. Microscopical examination of the urine revealed nothing of interest. His heart and lungs appeared normal. No malarial organisms were detected in blood films and no enteric organisms were recovered from the fæces or by blood culture. Agglutination tests were carried out: (i) against *Bacillus typhosus, Bacillus paratyphosus A* and *B* and *Bacillus proteus* X19 with negative results; (ii) against *Brucella abortus* and *Brucella melitensis* with strongly positive results, agglutination occurring with the former organism up to a serum dilution of one in 2,560, with the latter organism to one in 1,280. A blood count done by Dr. McLean showed 3,500,000 red blood corpuscles per cubic millimetre, white blood corpuscles 6,900 and hæmoglobin 72%. A differential count gave the following results:

Neutrophile polymorphs	28·5%
Eosinophile polymorphs	1·5%
Basophile polymorphs	0·5%
Lymphocytes	51·5%
Atypical lymphocytes	1·5%
Monocytes	9·5%
Metamyelocytes	7·0%

Blood culture on Huddleston's broth and agar has been attempted without success. Further efforts are being made in this direction.

The chief symptoms from which the patient has suffered since entering the hospital, have been fever, malaise, constipation, pains and swellings about the joints.

The temperature of the patient during his stay in hospital is indicated in Chart II.

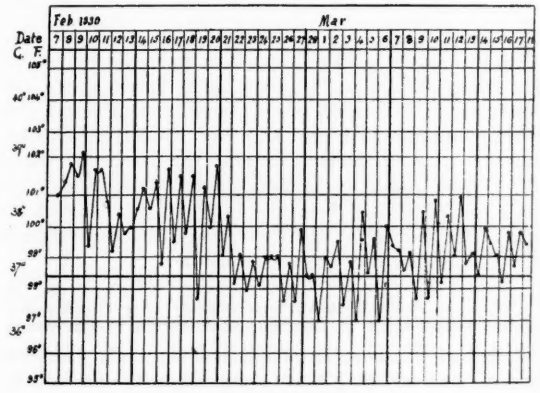

CHART II.

Undulant Fever Abroad.

Denmark.

The position in respect of undulant fever in Denmark has been set out by Dr. Madsen, Director of the State Serum Institute.[4] From his report we learn that of 2,500 samples of blood serum derived from 2,150 patients no less than 222 gave typical agglutination reactions at least in a dilution of one in 100 with *Bacillus abortus* of Bang. One hundred and seventy-two sera of these 2,150 patients gave a positive Widal reaction with *Bacillus typhosus* and 126 with *Bacillus paratyphosus B*, from which Madsen concludes that infection with *Bacillus abortus* is more frequent in Denmark than infection with *Bacillus typhosus*. In five of the cases agglutination was found against both *Bacillus typhosus* and *Bacillus abortus*. Apparently undulant fever in Denmark has become a disease of national importance.

In 27 of the 222 cases giving a positive agglutination with *Bacillus abortus* blood cultures were carried out with eighteen successes. Among 3,175 sera sent in to the Serum Institute, Copenhagen, for Wassermann test, only thirteen reacted in the complement fixation test with *Bacillus abortus*. They reacted only in low dilution. The number of these sera that gave positive agglutination reactions is not recorded, so that they are not a very good control.

Fifty control sera taken from a district in Greenland where no cattle existed are all said to have given negative results, but it is not stated whether this refers to agglutination or complement fixation tests or both.

There was no instance of more than one case occurring in the same family and there was no grouping of the cases in any particular part of the country, except such grouping as could be accounted for by increased density of population in the district.

Males were much more frequently affected than females, especially in the rural districts. The bulk of the rural males affected with the disease were working in close contact with cattle infected with *Bacillus abortus*.

The usual age period for the infection was fifteen to forty in males. No case was discovered in children under eight years of age. No age group was especially liable to the disease in the case of females. The mortality was only about 2%.

Madsen does not appear to consider that goats play any part in the spread of this disease in Denmark and none of the cases dealt with was infected by goats or goat's milk as far as could be judged.

Madsen speaks of the close relationship, if not identity, of *Bacillus melitensis* and *Bacillus abortus* of Bang.

The United States of America.

This subject has been dealt with very exhaustively by Hardy.[5] In 1924-1925, in Texas, Arizona and New Mexico ten cases of undulant fever were recognized. They were all believed to be of caprine origin. The importance of the subject had grown so enormously that in 1928 and the first five months of 1929 more than one thousand cases of undulant fever were diagnosed in the United States.

Apparently the infection in the States is derived from three sources—goats, pigs and almost certainly bovines. The relationship of these various forms to one another is as yet undecided. Evans in 1918

first pointed out the similarity of the *Micrococcus melitensis* and the *Bacillus abortus*. The porcine variety was isolated in 1914. In the United States twenty-one out of twenty-eight strains from the human subject were found to be of the porcine type, while apparently a smaller number were of the bovine type.

In Hardy's opinion goat's milk is more likely to spread infection than cow's milk, because of the larger number of organisms in the goat's milk. Having regard, however, to the frequency of laboratory infections with *Micrococcus melitensis*, this organism is probably more virulent for man. In the United States, direct contact with living infected animals or with infected carcasses has been found to be an important factor in the spread of the disease.

Hardy interprets the agglutination reaction as follows: If the serum has a titre of one in 40 it is doubtful whether it indicates infection or not, a titre of one in 80 he looks upon as weakly positive, one in 160 as positive and one in 320 as strongly positive. This interpretation does not differ widely from that of Madsen in Denmark who takes agglutination at a dilution of one in 100 as positive evidence of infection.

Bierring[6] studied 150 cases of undulant fever in Iowa. They showed a 2% mortality and the agglutination titre varied from one in 40 to one in 10,000. Three fatal cases showed agglutination titres respectively of one in 80, one in 160 and one in 640.

Theobald Smith[7] has examined twenty-three strains of organisms of the brucella group isolated from the human subject. Eighteen of these were isolated in the United States. He has classified them according to their pathogenicity for guinea-pigs, recognizing four grades of pathogenicity:

1. Strains causing no effect whatever on the guinea-pig. The strains cannot be recovered from the animals even if the entire spleen be subcultured.

2. Strains causing enlargement and congestion of the spleen, slight enlargement of lymph glands and atrophy of the testicles.

3. Strains causing some small necrotic foci in spleen and lymph glands.

4. Strains causing relatively large or numerous small necrotic suppurative foci.

The bulk of the strains he examined fell into the fourth group. The degree of virulence this group showed was so different from that shown by genuine bovine strains, that he suggests they may have been strains modified by passage through swine.

Smith examined three of the strains recently isolated in Denmark and the evidence that he obtained from the consideration of two of these strains suggested they were of bovine type. The third strain was avirulent for guinea-pigs and he considered it possibly a saprophytized strain of *Bacillus abortus* derived, perhaps, from vaccination.

Smith was not able to differentiate between his various strains by the absorption process. In this matter his results differ from those of Evans. He groups his strains according to virulence in the guinea-pig, but the precise significance of these various degrees of virulence is left in doubt.

In one year from January 26 to March 28 twenty-four cases of undulant fever were found in the State of New York. Twenty-three of these infections were believed to be due to *Brucella abortus* and one to *Brucella melitensis*.

South Africa.

Ross[3] described eight strains obtained from cases of undulant fever in Rhodesia. He believed that six of these strains were *Brucella abortus* and two were *Brucella para-abortus*. He came to these conclusions after examining nine brucella strains collected from various countries. He showed that these strains could be differentiated into two groups by agglutination alone, one group containing *Brucella melitensis* and *Brucella abortus* organisms and the other group containing the *Brucella para-melitensis* and *Brucella para-abortus*. Each member of these pairs could be distinguished from its fellow by agglutinin absorption tests. He suggests that these para strains are more closely related to each other than *Brucella para-abortus* is to *Brucella abortus*. He considers the para strains are probably rough types and the original *melitensis* and *abortus* strains are smooth types in the current sense of the terms. The para strains on injection produced only sera of low agglutinating titre. Agglutination reactions with them were characterized by sedimentary rather than granular deposits, while *Bacillus melitensis* and *Bacillus abortus* gave granular agglutination with their respective sera. The absorption results described by Ross were extremely clear cut.

England.

Malta fever in England has been described by Theodore Thompson[8] and others. Thompson's case occurred in a farmer whose herd was affected with contagious abortion and who had used living Bang's bacillus as a protective vaccine for his herd. Many other cases have been described in England. The bulk of them have been diagnosed by means of the agglutination test. Cultivation of the organism in blood cultures has been exceptional.

The Use of Bacillus Abortus (Bang) as a Vaccine in the Prophylaxis of Contagious Abortion of Bovines.

Contagious abortion of bovines is widely prevalent and it was natural that when Bang discovered the causal organism of the disease, its use as a prophylactic vaccine should be attempted. The vaccine was used in two forms, the dead and the living. It was found in practice that the dead organism gave a very small amount of protection against the disease in bovines, while extensive figures from various countries showed that the living vaccine, properly applied, reduced the abortion incidence in herds to one half or even less, as compared with the unvaccinated controls.

This caused the widespread use of the living vaccine which, for example, was supplied by the British Government in large quantities in England and by various other authorities in the United States of America, Denmark, Germany, France, South Africa and Australia. Protests against this policy arose on the ground that carriers in bovines might be artificially created by it and the disease itself on that account spread to uninfected herds. This consideration weighed with the Victorian veterinary authorities to such an extent that in July, 1923, an Order in Council was made prohibiting the use of the living vaccine, except by the special permission of the State veterinary authorities. Similar prohibitions were made in other States about the same time, so that the use of the living vaccine was abruptly stopped in Australia. Its use is still continued in England, Germany, Denmark, France and in many other countries.

This policy of the local veterinary authorities did not meet with universal approval, chiefly on the ground that if any large herd in the State were tested, some infected animal or animals would almost certainly be found in the series. That at least was the experience of the Commonwealth Serum Laboratories about the years 1922 and 1923.

The increasing prevalence of undulant fever in various countries raises the question of possible risk to man of this form of protection of bovines against contagious abortion. Professor Bang, in Copenhagen, is strongly of opinion that it does not represent any risk to man. In a personal letter, under date October 22, 1929, he answered a query on this subject in the following terms:

> As to the vaccine causing undulant fever, I can tell that in this country (Denmark) last year fifteen thousand animals were treated with living culture, most of them non-pregnant heifers, which is considered without risk to inject. We have in Denmark about 1·6 million milking cows. Undulant fever is just as frequent in districts where living culture has never been used as in districts where it is used.

He emphasizes also that the organisms used for living vaccines are of definitely diminished virulence as compared with the organisms freshly cultivated from cases of spontaneous abortion or from infected udders. He believes that the cases of undulant fever occurring in the country in Denmark are mostly caused by direct infection from the aborting cows, since the farmers generally do not drink much milk. This view is supported by the fact that undulant fever is more frequent in men than in women. It is the men who come into actual contact with the cows.

Whatever may be the part played by the living vaccine in the spread of undulant fever in other parts of the world, it seems to us it cannot possibly account for our Australian cases, since no living vaccine has been used for the last seven years and when it was used in Australia it was confined to Victoria. None was used in South Australia and our first patient apparently got his infection there.

Acknowledgement.

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References.

(1) H. E. Albiston: "The Occurrence of Brucella Abortus (Bang) in Market Milk and Its Relation to Disease in Man," THE MEDICAL JOURNAL OF AUSTRALIA, June 29, 1929, page 863.

(2) Alice C. Evans: "Further Studies on Bacterium Abortus and Related Bacteria: (ii) A Comparison of Bacterium Abortus with Bacterium Bronchisepticus and with the Organism which Causes Malta Fever," *Journal of Infectious Diseases*, Volume XXII, 1918, page 580.

(3) G. R. Ross: "Serological Study of Undulant Fever in Southern Rhodesia," *Journal of Hygiene*, October, 1927, Volume XXVI, page 403.

(4) T. Madsen: "Health Reports," League of Nations, May, 1928.

(5) A. V. Hardy: "Undulant Fever: Etiology, Epidemiology and Laboratory Diagnosis," *Journal of the American Medical Association*, Volume XCIII, September 21, 1929, page 891.

(6) W. L. Bierring: "Undulant Fever: Clinical Characteristics Based on Study of 150 Cases Observed in Iowa," *The Journal of the American Medical Association*, Volume XCIII, September 21, 1929, page 897.

(7) Theobald Smith: "Undulant Fever," *Medicine*, Volume VIII, Number 2, May, 1929, page 193.

(8) Theodore Thompson: "Bacillus Abortus Infection in Man," *The Lancet*, December 29, 1928, Volume CCXV, page 1335.

ORBITAL CELLULITIS IN CHILDREN.[1]

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THE subject of my paper is peculiarly suitable for the Pædiatric Section, as orbital cellulitis is much more frequent among children than adults; and my excuse for it, if such be needed, is that the condition as met with in children is not, so far as I am aware, adequately or concisely dealt with in text books. This paper not being written for the specialist, I may be pardoned if I lay emphasis on the gravity of the condition and dwell somewhat on its differential diagnosis.

Since orbital cellulitis may occur in children of a few weeks old, there is no age limit. The essential feature is proptosis of the eye associated with signs of inflammation. Proptosis alone is not sufficient for its diagnosis. Proptosis unassociated with inflammation may be caused by a cyst, a mucocele, a new growth or what not. I repeat therefore that not until one meets the combination of a congested eye and pink swollen lids with proptosis is the diagnosis established. Once the diagnosis is made, a grave prognosis should be given and the help of the specialist sought.

[1] Read at a meeting of the Pædiatric Section of the New South Wales Branch of the British Medical Association on April 11, 1930.

The diagnosis is not however always easy, as it is at times very difficult to be sure whether or not proptosis is present when chemosis of the conjunctiva and a swollen lid are the most prominent features. But it is on the existence of this sign that the diagnosis will depend. Proptosis is mechanically produced by congestion of, or exudation into, the orbital tissues behind the orbital fascia. This is a dense and well fitting septum separating the orbit proper from the conjunctival sac and the anterior segment and interior of the eyeball. It matters not how severe the infection of an eye, orbital cellulitis, with its cardinal symptom of proptosis, will not arise unless the orbital fascia is penetrated.

It must be clear then that orbital cellulitis must not be diagnosed because œdema of the eyelid is present or even cellulitis of the eyelid. The latter is often due to the spread of infection from a hordeolum or impetiginous sore in the neighbourhood. And œdema can often be traced to a cellulitis of the scalp or face, or dacryocystitis, and is then present merely by extension owing to the loose cellular structure of the lids.

Again, an intense purulent conjunctivitis, gonorrhœal or diphtheritic, may occasionally give rise to a pink and swollen lid; ocular diphtheria is frequently at first monocular. The diagnosis will be established by the presence of a profuse discharge or of a membrane on the everted lids. For the determination of the presence or absence of proptosis lid retractors are essential. These may be improvised by the use of two hairpins (if obtainable) bent over near the blunt end. If a local cause for the lid swelling, whether abscess, conjunctivitis, dacryocystitis or foreign body has been discovered, it will be treated on appropriate lines. If, however, none such is found and proptosis is present or strongly suspected, a grave view must be taken of the condition as it is one of orbital cellulitis.

Orbital cellulitis in children has, so far as my experience goes, three chief causes:

1. Skin infections such as impetigo, hordeolum, flybite, boils or any facial wound, with accidentally superadded virulent infection. This is more likely to be met with in older children. The temperature is generally very high; septic cavernous sinus thrombosis and general septicæmia with pneumonia are likely to supervene and the affection often ends fatally and rapidly. Fortunately such cases are rare. I have one or two in mind. The infection passes by the angular vein to the cavernous sinus.

2. Nasal sinus infection, the commonest cause. These cases are met with in young infants as well as in older children. Fever is not usually very high, if present, and the child is not very ill. The eye is pushed outwards as well as forwards. This is a strong diagnostic point. I will go so far as to say that if the eye is not displaced outwards, the infection is not nasal in origin. In these cases the services of the rhinologist should be sought, who will, if he thinks necessary, open up the antrum or ethmoidal cells. I believe the most usual condition is an ethmoiditis. If the proptosis is due only to œdema, it will gradually subside. If, however, pus has formed in the orbit, a counter opening between the nose and eye may be needed. Convalescence is slow but sure and the eye is not usually in jeopardy. I have seen a considerable number of examples of this type of cellulitis in the last fifteen years. They are sometimes associated with the exanthemata.

3. Osteomyelitis or periostitis of the orbit. Until about three years ago I had not seen such a case.

I was then asked to see a young infant with a high temperature and pink œdema of the eyelids of both eyes who was profoundly ill. There was no injection of the ocular conjunctiva. As both eyes were affected, it was difficult to say whether or not proptosis was present. I opined, however, that it would be by the morrow and that it was an early case of septic cavernous sinus thrombosis, that nothing could be done, but that my rhinological colleague should be asked if he concurred. He did concur. On the next day I saw the child again. I still found no ocular injection to speak of and no definite proptosis, though the lids were more pink and swollen. I realized then that I had made an erroneous diagnosis, deceived by the binocular affection, which will generally point to a common origin. I asked that my colleague should see the child again. He found an osteomyelitis of the lower rims of both orbits. Incisions were made over the malar bones and the child recovered.

This was not a typical case, however, as the condition was binocular, surely a rare occurrence. Furthermore, although there was osteomyelitis of the malar bones, the lesion was anterior to the attachment of the orbital fascia and therefore, though œdema of the lids was present, there was no orbital cellulitis.

I have since seen three cases of orbital cellulitis (monocular) due to osteomyelitis of the orbit and not apparently associated with or due to sinus infection. I am sure that these cases are metastatic. They have not been coincidental with a similar lesion in the long bones and epiphyses, though they are without doubt analogous. The child becomes very ill indeed, with a high and septic temperature. Pink œdema of the lids and definite proptosis are early signs. The eye is pushed forward without being pushed outwards. Presumably the eye might be pushed to some extent away from the side of origin of the infection, wherever it might be. But in the early stages at any rate, when the proptosis is merely due to œdema and not to formation of pus, there is no lateral displacement. This is in contradistinction from those of nasal origin. In the last three cases that I have seen, the point of maximal swelling and tenseness of tissues has been at the lower outer side of the orbit. Possibly a trauma may have been the exciting cause.

In the first case I was not called in for several days, as a tentative diagnosis of a nasal origin has been made and the rhinologist had explored the sinuses with an indecisive result and with no improvement in the patient. I found the outer lower orbital margin tense and swollen and incised it here, getting pus freely. The boy ran a high temperature for many weeks and was very ill. Although there was free evacuation of pus the orbital œdema and

proptosis persisted and in spite of all efforts the cornea sloughed and the eye had to be eviscerated. Subsequently he developed a mild osteomyelitis of the facial surface of the malar bone.

The next case was that of a young infant, seen in private practice, with proptosis and similar symptoms. I temporised for a day and then opened the orbit at the outer angle, evacuating pus. Again, the orbital œdema was so great and persistent that the cornea sloughed and the eye had to be eviscerated, though the child recovered. Sequestra formed for a long time.

The third case was that of a boy of five with the same symptoms of high temperature, severe illness, inflammatory central proptosis and the chief swelling at the lower outer orbital margin. He had been ill two or three days. This time, profiting by my experience, I opened the orbit within half an hour of seeing him, passed a sinus forceps through the orbital fascia down to the bony floor and rim. No pus was seen, but on the next day it appeared alongside the tube which had been left in. The wound was kept open, the proptosis went steadily down and the eye was never in danger. He had a septic temperature for several weeks and had some meningeal symptoms, which however subsided on lumbar puncture. *Staphylococcus aureus* was found in the blood and urine, so that there was evidently a very generalized infection. He, later, had an attack of osteomyelitis of the tibia.

To recapitulate, apart from orbital cellulitis due to facial infection, in which the orbital condition is only an incident in a fatal septicæmia, there are two main causes. Firstly, and the most common, nasal sinus infection, with outward displacement of the proptosed eye and not very severe general illness. Here the rhinologist can give most help and if he is called in early incision into the orbit will probably be avoided. Secondly, metastatic osteomyelitis or periostitis of the orbital walls, with severe illness and temperature and central proptosis. The lesson I have learned from these latter cases, and which I want to impress on you, is that early incision (as in the analogous disease of the long bones and epiphyses) is essential if the eye is to be saved. When once the orbital tissues are thoroughly infiltrated and œdematous resolution seems to be difficult or impossible before the cornea has sloughed from interference with its circulation, as well as from exposure. Our aim should be to prevent this phlegmonous condition by prompt incision down to the bones involved. Hence the importance of early and accurate diagnosis. To assist you in this is my reason for bringing the results of my experience before you. In the presence of a pink and swollen eyelid it is the part of the general practitioner to realize the significance of proptosis, if this exists. In its absence he may temporise; in its presence he should act with decision. No case of inflammatory exophthalmos should be regarded lightly.

But although I have dwelt in more detail on the signs and symptoms of the type of orbital cellulitis due to bone infection by the blood stream, that is not because it is the commonest (which it is not), but because it is the most grave and demands the most promptitude in its treatment. According to Roemer more than 60% of cases are due to nasal sinus infection. He is referring to adults and I think that this applies much more so to children owing to the thinness of the orbital walls. In my own practice orbital cellulitis in adults is exceedingly rare, whereas in children it is at least not uncommon and this I think is the explanation.

My aim in this short paper is twofold: one, to emphasize the importance of proptosis when associated with inflammation, and the other, to attempt to make clear the difference in signs and symptoms of the two main types of orbital cellulitis and the difference in outlook and treatment which each demands.

BACILLARY INFECTIONS OF THE URINARY TRACT: A BACTERIOLOGICAL STUDY.

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A RECENT study of the subject of phosphaturia carried out in this laboratory by Laidley[1] has demonstrated the fact that a large number of organisms, both bacilli and cocci, occurring in urinary tract infections, a much larger number than was previously realized, has the power of fermenting urea, with the production of ammonium carbonate and consequently phosphaturia with ammoniacal urine. Out of a series of one hundred and eighty Gram-negative bacilli 23·3% were able to split urea and fell into five fairly well defined groups, only one of which had been described previously.

It was felt that further routine work along the same lines would be of value, both in affording confirmation of the results already obtained and in supplying a larger series of organisms for the purpose of permanently classifying these urea-splitting bacilli, for the most part undescribed till the recent investigation mentioned above. The cocci have already received attention and are being reported on elsewhere.[2]

TECHNIQUE.

A series of two hundred consecutive bacilli isolated from urinary tract infections was examined, notes being kept regarding the source of each culture in order to avoid duplication of results, no two cultures, therefore, being derived from the same patient. All cultures were inoculated into the urea medium which was made up as already described by Laidley, with the exception that the concentration of urea therein was increased from 0·75% to 1·0%. In addition each culture was partly differentiated by inoculation into gelatin, litmus milk, glucose broth (for carbinol), peptone water (for indol), nitrate broth (for nitrites), lactose, glucose, maltose, mannite, saccharose and dulcite. All the urea-splitters were further inoculated into the rest of the bacteriological sugars. In addition to those employed in the previous investigation, amygdalin, mannose and rhamnose were also used. Quantitative estimations of the amounts of urea split were not undertaken, it having already been shown that the criteria

afforded by the qualitative method are quite reliable. Urea-splitting properties are described below as "++" or "+" according to the final colour of the medium (this contains British Drug Houses "universal indicator," which demonstrates the degree of alkalinity produced by the formation of ammonia from the urea as a result of the activity of the organism undergoing investigation). Where at the end of four days' incubation the medium showed a deep blue colour with a violet tinge, indicating a pH of about 8·7, and the addition of a few drops of an alcoholic solution of phenolphthalein produced a strong red colouration, a maximum amount of urea was assumed to have been split and the organism in question was listed as a strong urea-splitter ("++"). Where, on the other hand, the medium was a greenish blue, indicating a pH of about 8·5 and addition of phenolphthalein produced a weak red colouration only, the organism was listed as a weak urea-splitter ("+"). Where the colour of the medium remained green, indicating a pH of 8·0 as before inoculation and phenolphthalein produced no colouration, no urea was assumed to have been split.

In addition to the above work a similar study was made on a series of fifty organisms isolated from the intestinal tract in order to ascertain which, if any, of these had urea-splitting properties and to establish their identity, if possible, with the organisms possessing this property met with in urinary infections.

RESULTS.

Organisms Isolated from Urinary Infections.

Proportion of Urea-Splitting Organisms.

Out of the two hundred organisms used in this investigation, fifty-six were found to split urea to a greater or less extent, that is, 28%, a rather higher figure than the 23·3% found by Laidley. A larger number of organisms of the *proteus* group, however, which are well known urea-splitters, was met with in the present series. Excluding organisms of this group from both series, we find that in Laidley's series 27 out of 165 split urea, that is, 17%, while in the present investigation 35 out of 179 had this property, that is, 19·5%, which figures correspond more closely than those provided by the more superficial comparison.

The Non-Urea-Splitting Organisms.

These organisms, 144 in number, were classified by reference to Bergey[3] as follows:

Escherichia coli	54
Escherichia communior	36
Aerobacter aerogenes	6
Salmonella	7
Pseudomonas	1
Miscellaneous (unidentified)	40

The number of non-identified organisms is rather high. Eight of these, except for their production of carbinol, had the cultural characteristics of *Escherichia communior*, while a similar property prevented another one from being classified as *Escherichia coli*. Five other organisms did not produce carbinol, but their behaviour was otherwise typical of *Aerobacter aerogenes*. It may therefore be, as Laidley suggests, that rather too much stress is laid by Bergey on the presence or absence of carbinol formation in the classification of these organisms. Thirteen of the non-identified organisms formed a well defined group which could not be classified according to Bergey. They were motile organisms, not fermenting either saccharose or dulcite, but forming acid and gas in the other four sugars used. Gelatin was not liquefied, carbinol formation was absent, while indol and nitrites were both formed. They formed large whitish colonies with some spreading tendency and occurred also as normal inhabitants of the intestinal tract (see below). According to some other but older authorities these are the cultural reactions of the *Bacillus acidi lactici*, but Bergey's description of the organism which he lists as the *Escherichia acidi lactici*, is quite different. Nine of the non-identified organisms formed acid only in carbohydrate media, while the remaining four formed acid and gas in one or more of them, but defied classification.

It appears, therefore, as noted in the previous series, that the *Escherichia coli* and *communior*, the *Aerobacter aerogenes* and the *salmonella* and *pseudomonas* groups, as well as the well defined group which, according to some authorities, but not Bergey, may be the *Escherichia acidi lactici*, do not possess the property of fermenting urea.

The Urea-Splitting Organisms.

The urea-splitting organisms, fifty-six in number, could be grouped according to Laidley's scheme, with the addition of one type and two subtypes which did not occur in his series. They are listed in Table I. The key to the classification, slightly amended from that previously suggested, may be put forward as follows:

A. Form acid and gas in carbohydrates.
  1. Acid and gas in lactose.
    a. Acid and gas in dulcite.
      (i) Acid and gas in saccharose ..... Type A.
      (ii) No acid or gas in saccharose .... Type B.
    b. No acid or gas in dulcite.
      (i) Acid and gas in saccharose ..... Type C.
      (ii) No acid or gas in saccharose .... Type D.
  2. No acid or gas in lactose ..... Type E. (Type D, Laidley)

B. No gas, but only acid in carbohydrates .... Type F. (Type E, Laidley)

Type A which formed a fairly large proportion of the series, two of the four subtypes of which correspond to the two in Laidley's type A, includes nineteen organisms which fall into two main divisions according to the presence or absence of carbinol formation. Those that form carbinol can be separated as before into two subtypes (1 and 2), of which one does not liquefy gelatin and has weak urea-splitting properties, while the other liquefies gelatin and is a strong urea-splitter. There is a tendency to indol formation in both groups, absent

TABLE I.

Number of Subtype.	Serial Number of Organism.	Cultural Appearance.	Litmus Milk.	Motility.	Gelatin.	Carbinol.	Indol.	Nitrites.	Lactose.	Glucose.	Maltose.	Mannite.	Saccharose.	Dulcite.	Galactose.	Glycerin.	Xylose.	Adonite.	Lævulose.	Mannose.	Inosite.	Sorbite.	Erythrite.	Salicin.	Arabinose.	Raffinose.	Rhamnose.	Amygdalin.	Inulin.	Dextrin.	Starch.	Urea.
Type A.																																
1	3	Thick whitish non-spreading colonies	AC	+	–	+	–	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	AG	AG	AG	+
	88		AC	+	–	+	–	–	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	+
	29		AC	+	–	+	–	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	AG	AG	AG	+
	39		AC	+	–	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	–	AG	AG	AG	+
2	74	Proteus-like	AC	+	liq.	+	–	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	++
	185		AC	+	liq.	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	AG	AG	AG	++
	53		AC	+	liq.	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	–	AG	–	–	AG	–	AG	–	AG	–	+++
	181		AC	+	liq.	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	–	AG	–	–	AG	AG	AG	–	–	AG	–	++
3	24	Thick whitish non-spreading colonies	AC	+	–	–	+	–	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	–	AG	–	–	AG	AG	AG	AG	–	AG	AG	AG	++
	42		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	–	AG	–	–	AG	AG	AG	AG	–	–	AG	–	+
	108		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	–	AG	AG	AG	AG	–	–	–	–	+
	186		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	–	AG	AG	AG	AG	A	–	AG	AG	++
	197		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	–	AG	–	–	AG	AG	AG	AG	–	–	–	–	+
4	47	Proteus-like	AC	+	liq.	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	–	AG	AG	AG	AG	A	–	AG	AG	++
	12		AC	+	liq.	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	–	–	AG	AG	AG	–	A	AG	AG	AG	+++
	48		AC	+	liq.	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	–	AG	AG	AG	–	A	–	AG	AG	+++
	113		AC	+	liq.	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	–	–	AG	AG	AG	AG	A	AG	AG	AG	+++
	115		AC	+	liq.	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	–	–	AG	AG	AG	AG	A	AG	AG	AG	+++
	184		AC	+	liq.	–	+	–	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	–	–	AG	A	–	AG	–	++
Type B.																																
—	128	Whitish colonies, with slight spreading tendency	AC	+	–	–	+	+	AG	AG	AG	AG	–	AG	AG	AG	–	–	AG	AG	–	AG	–	AG	AG	–	AG	–	–	–	–	+
Type C.																																
1	125	Proteus-like	AC	+	liq.	–	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	–	AG	AG	–	–	–	AG	–	–	–	A	–	AG	AG	++
	150		AC	+	liq.	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	+
	153		AC	+	liq.	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	–	AG	AG	–	AG	–	AG	AG	AG	AG	A	–	AG	–	++
	164		AC	+	liq.	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	+
	176		AC	+	liq.	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	–	–	AG	–	++
2	85	Thick whitish non-spreading colonies	AC	+	–	–	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	++
	174		AC	+	–	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	–	+
Type D.																																
—	40	Whitish colonies, with some spreading tendency	AC	+	liq.	–	+	+	AG	AG	AG	AG	–	–	AG	AG	AG	–	AG	AG	–	–	–	–	–	–	–	–	–	–	–	++
	102		AC	+	liq.	–	+	+	AG	AG	AG	AG	–	–	AG	AG	AG	–	AG	AG	–	AG	–	AG	AG	AG	AG	–	–	–	AG	++
	121		AC	+	–	–	+	+	AG	AG	AG	AG	–	–	AG	AG	AG	–	AG	AG	–	AG	–	AG	AG	–	–	–	–	–	AG	+
	146		AC	+	–	–	+	+	AG	AG	AG	AG	–	–	AG	AG	AG	–	AG	AG	–	AG	–	–	AG	–	AG	–	–	–	–	++

A = Acid. AC = Acid and clot. AG = Acid and gas.

TABLE I (*Continued*).

Number of Subtype.	Serial Number of Organism.	Cultural Appearance.	Litmus Milk.	Motility.	Gelatin.	Carbinol.	Indol.	Nitrites.	Lactose.	Glucose.	Maltose.	Mannite.	Saccharose.	Dulcite.	Galactose.	Glycerin.	Xylose.	Adonite.	Levulose.	Mannose.	Inosite.	Sorbite.	Erythrite.	Salicin.	Arabinose.	Raffinose.	Rhamnose.	Amygdalin.	Inulin.	Dextrin.	Starch.	Urea.
														Type E.																	
1	41	Proteus-like	Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	++
	55		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	109		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	124		Alk. (peptnoized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	127		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	149		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	159		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	–	–	–	–	–	–	–	–	–	–	–	–	+++
	163		Alk. (peptonized)	+	liq.	+	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	171		Alk. (peptonized)	+	liq.	+	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	173		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	192		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	200		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	5		Alk. (peptonized)	+	liq.	+	+	+	–	AG	–	–	–	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	46		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	–	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	69		Alk. (peptonized)	+	liq.	+	–	+	–	AG	–	–	–	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	89		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	–	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	107		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	–	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
	199		Alk. (peptonized)	+	liq.	–	–	+	–	AG	–	–	–	–	AG	AG	AG	–	AG	–	–	–	–	–	–	–	–	–	–	–	+++
3	19	Proteus-like	Alk.	+	liq.	–	+	+	–	AG	AG	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	AG	–	–	–	–	–	–	++
	30		AC	+	liq.	–	+	+	–	AG	AG	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	AG	–	–	–	–	–	–	++
	31		A	+	liq.	–	+	+	–	AG	AG	–	AG	–	AG	AG	AG	–	AG	–	–	–	–	AG	–	–	–	–	–	–	++
														Type F.																	
—	27	Whitish spreading colonies	AC	+	–	–	–	+	A	A	A	A	A	–	A	A	–	A	A	A	–	A	–	A	–	–	A	A	–	A	–	++
	63		A	++	liq.	+	–	–	A	A	A	A	–	–	A	–	–	–	A	–	–	–	–	–	–	–	A	–	–	–	–	++
	86		A	++	–	–	++	+	A	A	A	A	–	A	A	–	A	–	A	–	–	A	–	–	A	–	–	–	–	–	–	+
	155		Alk.	+	–	–	+	+	–	A	–	A	–	–	A	–	–	A	A	A	A	–	–	–	A	–	–	–	–	–	–	++

A = Acid. AC = Acid and clot. AG = Acid and gas.

TABLE II.

Number of Subtype.	Serial Number of Organism.	Cultural Appearance.	Litmus Milk.	Motility.	Gelatin.	Carbinol.	Indol.	Nitrites.	Lactose.	Glucose.	Maltose.	Mannite.	Saccharose.	Dulcite.	Galactose.	Glycerin.	Xylose.	Adonite.	Levulose.	Mannose.	Inosite.	Sorbite.	Erythrite.	Salicin.	Arabinose.	Raffinose.	Rhamnose.	Amygdalin.	Inulin.	Dextrin.	Starch.	Urea.
														Type A.																	
1	A12	Non-spreading whitish colonies	AC	+	–	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	–	AG	AG	AG	AG	A	AG	AG	AG	+
	A31		AC	+	–	+	–	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	+
2	A36	Proteus-like	AC	+	liq.	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	–	AG	AG	AG	AG	A	–	AG	–	++
	A43		AC	+	liq.	+	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	AG	AG	AG	++
3	A11	Non-spreading whitish colonies	AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	–	AG	AG	AG	AG	–	–	–	–	++
	A33		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	–	AG	–	AG	AG	AG	AG	–	–	–	–	++
	A35		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	–	–	AG	AG	++
	A38		AC	+	–	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	–	AG	AG	AG	AG	–	–	–	–	++
	A49		AC	+	–	–	–	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	++
	A50		AC	+	–	–	–	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	++
4	A22	Proteus-like	AC	+	liq.	–	+	+	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	++
														Type B.																	
—	A32	Non-spreading whitish colonies	AC	+	–	–	+	+	AG	AG	AG	AG	–	AG	AG	AG	AG	AG	AG	AG	–	AG	–	AG	AG	–	AG	–	–	–	–	+
														Type C.																	
2	A42	Non-spreading whitish colonies	AC	+	–	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	–	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	+
	A44		AC	+	–	+	–	+	AG	AG	AG	AG	AG	–	AG	AG	AG	–	AG	AG	AG	AG	–	AG	AG	AG	AG	A	–	AG	AG	+

A = Acid. AC = Acid and clot. AG = Acid and gas.

in the previous series, while the sugar reactions are rather more variable, notably in the frequency with which inulin, dextrin and starch are fermented. Fundamentally, however, the organisms in these two subtypes are very similar in the two series.

Subtypes 3 and 4 have not been previously described. Carbinol formation is absent in both. Subtype 3 contains five members, of which three were weak urea-splitters, while two had this property more strongly developed. Gelatin is not liquefied, indol is formed and four of the five organisms formed nitrites. This subtype is very similar in cultural appearances and reactions to the *Escherichia communior*, differing from it only in its urea-splitting properties.

Subtype 4 contains six members and differs from Subtype 3 in that all are strong urea-splitters and liquefy gelatin, while the cultural appearance is different, the growth being of a spreading character very similar to that seen in the *proteus* group.

Of Type B only one representative was found, which differed from the three in Laidley's Type B in being only a weak urea-splitter and in failing to liquefy gelatin. These differences should be sufficient to justify the distinction of two subtypes, of which one, described by Laidley, is a strong urea-splitter and gelatin-liquefier, while the sole representative of the second is the organism just described.

Type C includes seven members. Five of these are strong urea-splitters and liquefy gelatin, corresponding to Subtype 1, whose characteristics they reproduce with sufficient accuracy, though the sugar reactions show some variation. Carbinol formation is present in four out of the five members, but the organism differs from the *Aerobacter aerogenes* in its urea-splitting and gelatin liquefying properties and also in cultural appearance. The remaining two members which do not liquefy gelatin, correspond to Subtype 3, though again the sugar reactions show some variation.

Four members of Type D, not encountered in the previous series, were found. Three of these were strong urea-splitters, of which two only liquefied gelatin. These organisms resemble fairly closely the group previously described, non-identifiable by Bergey's classification, but according to other authorities corresponding to the *Bacillus acidi lactici*. They differ, however, in the presence of urea-splitting properties and in two cases there is the further difference provided by gelatin liquefaction.

Of Type E (Laidley's Type D) twenty-one members were present. They fall into the *proteus* group of organisms and are all strong urea-splitters and gelatin liquefiers. Subtype 1, containing eighteen members, is identical with the subtype previously described except for occasional carbinol formation and the formation in one instance of indol. The remaining three members, though their cultural reactions in litmus milk differ, are most conveniently placed in Subtype 3, all of whose other characteristics they possess.

Of Type F (Laidley's Type E) four representatives appear. They form acid in the carbohydrate media, but no gas. Three of the four are strong urea-splitters. Their cultural characteristics are in no way consistent, the only constant properties, apart from urea-splitting power, being their motility and the absence of gas formation.

Organisms Isolated from the Intestinal Tract.

Fifty organisms isolated from the intestinal tract were examined, of which fourteen were found to have the power of splitting urea to a greater or less extent. This is a rather high proportion compared with that already found in urinary tract infections, particularly as no members of the *proteus* group were encountered, but the series is not a large one, as its study was not commenced till that of the other series of organisms was nearing completion.

The non-urea-splitters were classified by reference to Bergey as follows:

Escherichia coli	4
Escherichia communior	15
Aerobacter aerogenes	1
Eberthella typhi	1
Unidentified	15

As before, the non-identified organisms are present in somewhat large proportion, but most of them showed similar characteristics to those of the last series discussed. Thus four of them, but for the presence of carbinol formation, had the reactions of *Escherichia communior*, while two did not form carbinol, but otherwise corresponded to *Aerobacter aerogenes*. Six belonged to the unknown group previously described, unidentifiable by Bergey, but corresponding to the *Bacillus acidi lactici* of other authorities, while the remaining three were quite unclassifiable.

The urea-splitters, fourteen in number, are listed in Table II. Type A forms by far the majority and falls into the same four subgroups as previously described, which correspond fairly accurately in the two series. One representative of Type B is present and two of Type C, Subgroup 2. Organisms corresponding to the other types described in connexion with the series isolated from the urinary trace were not found, but since, as previously pointed out, the series examined is quite small, it is at least highly probable that further routine work would similarly establish their presence in the intestinal tract.

Discussion.

Comparison of Present Study with Previous Work.

Confirmation has been afforded of the findings already obtained as regards the comparative numbers and the classification of the urea-splitting bacilli which infect the urinary tract, and their intestinal origin has been sufficiently well demonstrated. The conclusions drawn by Laidley are borne out, with the exception that, although the great majority of the strong urea-splitters liquefy gelatin, this property is not a constant feature of the group. In the present series it was absent in

nine organisms out of forty-seven. It is possible that the reason for this may be found in the fact that the qualitative method used in this investigation is less accurate than the quantitative technique previously employed; in the present series, though the feeble urea-splitters were fairly accurately indicated, no idea could be formed of the relative amounts of ammonium carbonate produced by the more strongly urea-splitting organisms, owing to the constant alkalinity of this salt in solution, which does not exceed pH 8·8. It is thus possible that the nine non-gelatin-liquefiers, while strong urea-splitters as indicated by the colour of the medium, might have been shown by quantitative estimations to be comparatively weak ones, though not quite so weak as those in which this quality, though present, was at a minimum.

Urea-Splitting Powers as an Inherent Characteristic of the Organism.

The suggestion might be put forward that in view of the superficial resemblance some of the lactose-fermenting urea-splitters bear to other well known organisms not having this property, the power to split urea might be gradually acquired by such organisms as the latter during the course of a chronic infection of the urinary tract. Against this are the observations made in the present investigation that in numerous cases non-urea-splitting bacilli were isolated from infections of long standing, whilst in other instances strong urea-splitters were isolated from acute lesions. The strongest argument against the hypothesis, however, is the fact that organisms possessing strong urea-splitting properties were isolated from the intestinal tract, never having been the cause of a urinary infection, and that these organisms fell into the same groups as did those isolated from the urinary tract. The only possible inference that can be drawn from this is that the bacilli which give rise to urinary infections with ammoniacal urine, are certain definite urea-splitting strains which have their normal habitat in the alimentary canal.

Classification of Urea-Splitting Bacilli.

Sufficient work has been carried out in this laboratory to enable a fairly definite classification of these organisms to be made. Three hundred and eighty different strains of Gram-negative coliform bacilli have been examined, of which the ninety-eight urea-splitting bacilli have been fully differentiated, described and grouped. The remainder of the organisms examined, the non-urea-splitters, have been differentiated to an extent sufficient for fairly accurate recognition and it has been definitely established that certain well known organisms, that is, the *Escherichia coli* and *communior*, and others mentioned in the text, are not urea-splitters. As regards the urea-splitters, there are six main groups, of which four (A, B, C, D) bear some superficial appearance, greater or less, to organisms well known, while the *proteus* group is already well recognized and a definite bacteriological entity.

With regard to the lactose fermenters, there are two alternatives in their classification. The first is to regard the presence of urea-splitting properties as the fundamental characteristic of the group, while the second is to distribute the various types and subtypes over the range of the organisms already known which they most closely resemble (though the resemblance is in many caess anything but close), regarding each strain as a urea-splitting variation of a non-urea-splitting organism.

In view of the definite differences that exist between the urea splitters of types A, B, C, D and the non-urea-splitters which some of them superficially resemble, and, further, taking into consideration the fact that infections by urea-splitting bacteria, regarded from the clinical standpoint, are more or less a separate entity, the more rational procedure of the two would be to regard these four types mentioned as a separate bacteriological group, of which the presence of urea-splitting properties is the fundamental characteristic. Type E, the *proteus* group of organisms, is already allotted a definite place in the classification of the pathogenic microorganisms, while Type F, which forms acid only in the carbohydrate media, would seem to be regarded most conveniently as a variety of *eberthella*.

The Clinical Significance of Urea-Splitting Infections.

In many cases infections occur with urea-splitting organisms without the clinical signs of phosphaturia and ammoniacal urine. The commonest conditions in which these features occur, are those associated with obstructive lesions anywhere in the urinary tract. Where the bladder is emptied completely at each act of micturition, little time is available for much urea-splitting to take place and each time urine is passed the infecting organisms have, as it were, to start all over again. On the other hand, the constant presence of residual urine in the bladder makes conditions ideal for the production of ammoniacal urine and it is under these conditions that this change is most commonly met with. With a gross infection by organisms having very strong urea-splitting powers, definite phosphaturia and ammoniacal urine are occasionally seen in the absence of urinary obstruction, but the proportion of such patients with urea-splitting infections who show these features, is small. On the other hand, where residual urine is present, infection with a urea-splitter would almost certainly give rise to the characteristic clinical features in 100% of cases.

Similarly, when residual urine occurs in the renal pelvis with or without hydronephrosis due to the presence of ureteral calculus or stricture, ammoniacal fermentation with deposition of phosphates will precede these exactly as in the bladder, predisposing to the formation of calculi and to their steady increase in size.

With regard to therapy, the commonly accepted teaching is that in urinary infections with acid urine, the urine should be rendered alkaline and *vice versa*, with the object of inhibiting the growth

of the organism by means of the altered pH of the urine. The value of this procedure is well known in the presence of infections accompanied by acid urine. With a urea-splitting infection, however, in the first place it is extremely doubtful whether the urine could be ever rendered acid and, secondly, assuming that this could be done, it is not likely, in view of the fact that the organism at the onset of the infection had sufficient vitality to thrive in an acid urine to the extent of converting it into a strongly alkaline one, that its growth would be much inhibited. There would seem to be no reason in such infections to depart from the basic principles used in all urinary infections, namely, rest and copious fluids in acute infections, local treatment and promotion of free drainage in cases of longer standing.

CONCLUSIONS.

1. A very appreciable proportion of bacilli occurring in urinary tract infections has the power of splitting urea with the production of ammonium carbonate.
2. These organisms fall into well defined groups, most of which are not recognized among the generally accepted methods of classification.
3. A scheme of classification is given corresponding, with additions, to that already worked out in this laboratory by Laidley.
4. The organisms concerned are normal inhabitants of the intestinal tract.
5. The urea-splitting properties of these organisms become of clinical significance in the presence of residual urine or urinary calculus. Otherwise they are only of importance when very strongly developed.
6. Treatment of infections due to urea-splitting bacteria is that of urinary infections in general.

ACKNOWLEDGEMENT.

I am indebted to the Pathological Department of the Royal Prince Alfred Hospital for permission to make use of their urinary cultures for the purpose of this investigation and for making up many of the media used, and wish to record my appreciation of the unfailing assistance rendered by them at all times.

REFERENCES.

(1) J. W. S. Laidley: "Phosphaturia," THE MEDICAL JOURNAL OF AUSTRALIA, March 8, 1930, page 305.
(2) M. S. S. Earlam: "Urea-Splitting Staphylococci in Urinary Tract Infections." In the press.
(3) Bergey: "Manual of Determinative Bacteriology," 1925.

"SOMNIFAINE" ("SOMNIFENE"): ITS USE AS A PRELIMINARY TO ANÆSTHESIA.

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"SOMNIFAINE" is a solution of diethyl barbituric acid and allyl-isopropyl barbituric acid, containing of each 0·1 gramme per cubic centimetre in the form of the diethylamine salt. It is issued in two forms: (i) Ampoules of two cubic centimetres each for intravenous and intramuscular injection, (ii) bottles of twelve cubic centimetres for oral administration.

My attention was drawn to this drug by an article by J. H. Fjelde in *Anæsthesia and Analgesia*, January to February, 1929. To date the drug has been used intravenously in seventeen cases and orally in three. This report is based on these cases.

Intravenous Administration.

The results obtained by intravenous administration are set out in Table I.

With the exception of Case 12 there has been perfect freedom from any unpleasant effect, either before or after operation.

Patient XII was extremely unsuitable for operation. He had had asthma for twenty years. There was gross emphysema with a large heart whose borders could not be accurately defined owing to the emphysema. The serious risk was discussed with him, but he insisted on undergoing surgical treatment for relief of his cholelithiasis. He did not regain consciousness, but remained in a semicomatose and somewhat restless condition for ten hours after operation, when he died. Unfortunately, we were unable to obtain a *post mortem* examination.

With the exception of patients I and V, all of our patients arrived in the anæsthetizing room either very drowsy and care-free or actually asleep.

Patient I was a highly strung young girl, separated from her mother for the first time in strange hospital surroundings. She wept with ease and arrived in the anæsthetizing room weeping, but drowsy. After six breaths of anæsthetic she settled down into an extremely quiet condition of anæsthesia and remained so throughout the operation. In this case I progressively reduced the percentage of the ethylene and increased that of the oxygen in the mixture until for the last fifteen minutes of the operation anæsthesia was maintained with ethylene twenty parts, oxygen thirty, the safety gas machine being used.

In Case V there was little apparent effect from the injection, the patient was wide awake and talked vivaciously; but, in spite of this, she had no fear of the approaching operation. Induction and maintenance were quiet and easy.

Case IV was a great success. This patient had had her abdomen opened on a previous occasion and was very sick after the anæsthetic and dreaded the present one. She arrived in the anæsthetizing room sound asleep and the anæsthetization was easy, ethylene and oxygen being given with a little ether during the exploration. She dozed all the afternoon and had a very good first night, requiring no sedative in the first twenty-four hours. Next day she commented on the difference to the previous operation. There was no postoperative vomiting.

In Case III blood pressure readings were taken at five minute intervals after the injection. There was a drop of ten millimetres of mercury in ten minutes with recovery to normal in fifteen minutes.

The intravenous injection of "Somnifaine" should be given slowly, at no greater rate than one cubic centimetre per minute.

Should it be desired to counteract the drowsiness after operation, ephedrine, fifty milligrammes, and caffeine sodium benzoate, 0·18 gramme (three grains) should be injected, followed, if necessary, by two additional doses of the caffeine at hourly intervals.

Fjelde reports that there may be some postoperative restlessness which responds to an injection of morphine sulphate, 0·01 gramme (one-sixth of a grain). This restlessness has been more notice-

able after oral and intramuscular administration. So far none of our patients has given us any worry.

Another after effect which we have not yet encountered, is diplopia. In all Fjelde's patients diplopia subsided within eighteen hours.

Oral Administration.

In three cases "Somnifaine" has been administered orally.

CASE I. A male patient, aged twenty-three, suffered from inguinal hernia. Four cubic centimetres were given one hour before operation. Atropine sulphate, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), half an hour before. He was sound asleep and had to be wakened for his atropine. He then remained very drowsy until time of operation. Ethyl chloride and ether were used for induction and maintenance was rapid and easy and a very small quantity of ether was used. One and a half hours after operation he was awake but drowsy and complained of no discomfort. There was no vomiting.

CASE II. A male patient, aged forty-three years, suffered from inguinal hernia. "Somnifaine," four cubic centimetres, was given, followed in half an hour by 0·43 milligramme (one one-hundred-and-fiftieth of a grain) of atropine sulphate. Owing to difficulties in an earlier case, induction was not carried out until three hours after he had received his "Somnifaine." He slept soundly until half an hour before operation and was dozy for the remainder of the time. Ethyl chloride and ether were used, induction and maintenance being quiet and easy.

CASE III. A male patient, aged sixty-one years, suffered from renal calculus. When seen the day before operation he was excited and apprehensive of the next day's ordeal. "Somnifaine," four cubic centimetres, was given one and a half hours before and morphine sulphate, 0·01 gramme (one-sixth of a grain), and atropine sulphate, 0·43 milligramme (one one-hundred-and-fiftieth of a grain), one hour before operation. I found in this case that after the administration of the "Somnifaine" the patient's skin preparation had still to be completed; this was done whilst the patient was trying to follow instructions to try to sleep. In spite of this he walked into the theatre placid and care-free. Ethyl chloride and ether were used, induction and maintenance being quiet and easy. Four hours after operation he was still drowsy, but complained of pain which was relieved by morphine sulphate, 0·01 gramme (one-sixth of a grain). There was little vomiting.

From this brief experience of the drug it seems that "Somnifaine" is destined to be a useful addition to the armamentarium of the anæsthetist.

Number.	Age.	Sex.	Nature of Operation or Condition for which Operation was undertaken.	Systolic and Diastolic Blood Pressure in Millimetres of Mercury.	Weight.	Dose of "Somnifaine" in Cubic Centimetres.	Morphine.	Atropine.	Anæsthetic Used.	Condition on Arrival in Anæsthetizing Room.	Post-operative Vomiting.
1	17	F.	Adenoma of the thyreoid	130/85	29·8 kg. (84 lb.)	1·5	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Nervous, but drowsy	Very slight
2	61	F.	Exophthalmic goitre ..	168/82	50·4 kg. (112 lb.)	2·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Slight
3	15	F.	Appendicectomy ..	130/90	58·7 kg. (126 lb.)	2·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Nil
4	40	F.	Cholecystectomy ..	128/90	82·7 kg. (186 lb.)	4·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene, oxygen and ether	Asleep	Nil
5	23	F.	Exophthalmic goitre ..	138/88	58·7 kg. (126 lb.)	2·0	—	—	Ethylene and oxygen	Wide awake	Nil
6	26	F.	Adenoma of the thyreoid	110/80	60·75 kg. (135 lb.)	2·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Nil
7	52	F.	Cyst of breast	112/76	63·0 kg. (140 lb.)	4·0	—	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Once
8	43	F.	Radical amputation of breast	112/90	50·4 kg. (112 lb.)	3·0	—	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Asleep	Nil
9	36	F.	Cholecystectomy ..	150/100	60·3 kg. (134 lb.)	4·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene, oxygen and ether	Asleep	Nil
10	29	M.	Exploratory laparotomy	126/90	—	4·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene, oxygen and ether	Very sleepy	Slight
11	65	F.	Amputation of breast..	162/112	69·3 kg. (154 lb.)	4·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Nitrous oxide and oxygen	Asleep	Nil
12	63	M.	Cholecystectomy ..	125/60	63·0 kg. (140 lb.)	4·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene, oxygen and ether	Drowsy	Died ten hours after
13	50	F.	Amputation of breast..	85/55	61·2 kg. (136 lb.)	4·0	—	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Asleep	Several times
14	—	F.	Toxic goitre	—	—	2·0	—	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Slight
15	50	F.	Radical amputation of breast(No. 13 continued)			2·0		0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Very slight, less than previously
16	43	F.	Radical amputation of breast	112/90	50·4 kg. (112 lb.)	3·0	—	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Very slight
17	66	F.	Cholecystectomy ..	160/90	50·4 kg. (112 lb.)	2·0	0·01 gramme (1/6 of a grain)	0·43 mgm. (1/150 of a grain)	Ethylene and oxygen	Very sleepy	Slight

As a preparation for nervous patients it has acted excellently, as shown by the placid, care-free mental condition of the patients after injection.

As a preparation for local and spinal anæsthesia it should have a wide field, particularly as it is now known that the barbitone derivatives act as antidotes to poisoning by "Novocain" and allied local anæsthetic agents.

Another feature has been the ease of induction and I have the impression of using considerably less anæsthetic than with similar patients without "Somnifaine."

In the earlier cases morphine was given as a routine, but in the later ones the morphine is being left out, except in abdominal operations.

Reviews.

A BOOK ON MIDWIFERY FOR NURSES.

THE eighth edition of "A Short Practice of Midwifery for Nurses," by Henry Jellett, has now been published, a tribute to the popularity and excellence of this manual.[1] In this edition the general plan of the previous ones has been followed, but whereas in the last edition "The Aseptic Technique of Labour and the Puerperium" and "Suggestions to Expectant Mothers" were added as "Appendices," in the present edition they have been incorporated in the text, so that a new chapter devoted to antenatal care has been added and the chapters dealing with the preparations for labour and the management of the puerperium have been largely rewritten.

Minor alterations and additions have been made and the book generally revised. The teachings of Sir Truby King on infant feeding have been closely followed. Dr. Jellett's reputation as a sound teacher is so well known that it is a pleasure to see this new edition which brings the book right up to date.

We are of the opinion that this book will meet the needs of obstetric nurses. It is written in clear, simple language and a nice judgement is shown in the amount of description given for each procedure; sufficient is given to explain the reasons and yet the description is not too advanced for junior nurses to understand. Every aspect of obstetric work is discussed and useful appendices on subjects of interest and importance to obstetric nurses have been added. These greatly enhance the value of the book both as a text book and also as a book of reference after training is completed. We can unhesitatingly recommend this book to all obstetric nurses.

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM.

IT is obvious on reading "The Neuroses" that the author is a trained neurologist for whom the basic facts of neurological anatomy and physiology will not provide a satisfactory explanation to the phenomenon of the neuroses.[2] He insists that the psychogenesis of these disorders must be fully realized and a satisfactory and standardized terminology must take a more permanent place in our ordinary medical vocabulary.

In the first chapter the history of psychiatry from the earliest history of medicine is traced. The ancient Greeks recognized epilepsy, dementia and other psychoses as being definite disorders. This conception of mental diseases was lost in the mysticism of the middle ages, when all forms of disordered conduct were thought to be due to evil spirits. It is only comparatively recently that psychiatric disorders were again recognized as being diseases that might be prevented and that were amenable to treatment. We are only now in the transition stage of recognizing that psychological as well as physical mechanisms may give rise to symptoms of a definite disorder.

The development of modern psychopathology is dealt with, commencing with Charcot, Déjerene, Babinski and Wundt. The epoch-making classification of psychasthenia by Janet and the more modern dynamic theories of Freud, Jung, Adler and Bleuler are considered.

In the chapter on mental mechanism the theory of psychic determination, the physiological, somatic and the glandular approaches to the conception of the neuroses are all critically reviewed. Freud's theory of the conscious, preconscious and unconscious levels of mind and of sexual libido are detailed in a way that must appeal to readers of this book. He points out the rationale for Freud's terminology and predicts their general acceptance in medical training.

In the discussion of the ætiology of the neuroses a differentiation is made between the pathological and the physiological theories. In the former, symptoms arise due to organic alterations in cerebral neurones; in the latter it is assumed that there is a specific alteration in the normal physiological function of the brain—changes may occur at the synoptic junctions without any definite neuronic alterations.

The behavourist view, in which states of consciousness and unconsciousness are not recognized, thought and conduct being merely dependent upon highly organized conditioned reflexes, is given.

Disordered states of the glands of internal secretion are discussed in relation to their bearing on the production of neuroses.

The theory of suggestion and the social biological theories are also dealt with as a possible ætiological factor in relation to neuroses.

After weighing all the more materialistic theories in the balance and finding them all wanting, the author approaches the psychogenic theories with the same spirit of constructive criticism. It is obvious that the psychoanalytical approaches appeal to him from a scientific standpoint and the reader feels that the author has come to this conclusion only after mature and well balanced consideration.

Freud's classification of the neuroses is adhered to as being the least complicated to follow. Paraphrenia, schizophrenia, manic-depressive psychosis and paranoia are classified as the narcissistic neuroses. The actual neuroses, anxiety states, neurasthenia, hypochondriasis and traumatic neuroses, are those in which psychic trauma plays a part and in which actual secondary pathological changes may take place in the body.

A chapter is given on clinical manifestation of the neuroses with illustrative examples of each type of case. These are discussed and it is obvious that they are not selected "show" cases. The patients that do not respond to psychotherapy are discussed as fully as those in whom psychotherapy has succeeded. The author is of opinion that the modern trend of civilization is towards a higher psychic mode of expression in the production of neurosis symptoms. Instead of the great mass of hysterical paralysis, anæsthesias and general somatic symptoms that were formerly seen, the anxiety type of symptom complex is more in evidence.

[1] "A Short Practice of Midwifery for Nurses," by Henry Jellett, B.A., M.D. (Dublin), F.R.C.P.I.; Eighth Edition; 1929. London: J. and A. Churchill. Crown 8vo., pp. 510, with illustrations. Price: 8s. 6d. net.

[2] "The Neuroses," by Israel S. Wechsler, M.D.; 1929. Philadelphia: W. B. Saunders Company; Melbourne: James Little. Demy 8vo., pp. 330. Price: 20s. net.

In the discussion on diagnosis it is pointed out that there may be extreme difficulty in making a diagnosis and that unless a psychogenic cause can be discovered, one should have great hesitation in making a diagnosis of a neurosis. It is pointed out that organic diseases in their early stages simulate closely the symptoms of a neurosis and that a neurosis is by no means incompatible with an existing organic disease. The search for focal sepsis has become a fetish amongst certain physicians and the author points out that often a comparatively simple form of psychological examination may disclose a potent cause for symptoms, the patient thereby being saved much wearisome and expensive removal of problematical septic foci. On the other hand, the author stresses the importance of a most searching physical and neurological examination and willingly admits that septic foci may be a predisposing cause for neurotic symptoms. His point is that a better realization of the significance of psychological medicine by the medical profession may save the patient a considerable amount of unnecessary investigation.

From the point of view of prognosis an attempt should be made to put the patient's symptoms into a classification. This he realizes is often very difficult and the differentiation between a neurosis and an incipient psychosis may also be difficult.

As regards treatment, a grave note of warning is sounded against psychoanalysis as a therapeutic measure in unskilled hands and for unsuitable patients. The general management of cases of the neuroses is skilfully outlined and the common pitfalls are pointed out. Treatment by hypnosis and suggestion are suitable in certain conditions. A brief account of the psychoanalytical technique is given. The author advises the analyst to be analysed himself in order to remove any of his own resistances which may impede his treatment of a patient.

A chapter on the methods of history taking and general examination of a psychoneurotic patient is all embracing and is well worthy of any medical practitioner's close attention.

An outline of the Kuhlmann psychometric tests at different ages is contained in the last chapter.

The book is contained in 330 pages. The references and index are complete.

The general medical reader will have his interest in the neuroses stimulated and will be able to gain an insight into the modern conception of psychogenic disorders. This book is to be highly recommended for bringing these disorders into line with general medicine. Modern views are attractively put forward and the illustrative cases are profuse and well selected.

A COMPREHENSIVE HAND BOOK OF BACTERIOLOGY.

THERE has been recently published a book entitled "The Principles of Bacteriology and Immunity."[1] The joint authors are Professor W. W. C. Topley and Dr. G. S. Wilson, both of the University of London. They have based the work on their personal experience in post-graduate and undergraduate teaching. They have succeeded in writing a text book which will be of service not only to medical students, but to those of greater maturity who in ever-increasing numbers are making a serious study of bacteriology and of the problems of infection and resistance.

The book is divided into four parts. The first two in themselves form a modern manual of bacteriology; the third deals with the whole complex mechanism of infection and resistance; the fourth deals with the application of bacteriology to medicine and to hygiene. This fourth division is wholly admirable. Nowhere else have we seen the necessity for a liaison between the medical and veterinary bacteriologist stressed as it should be and as it is here. For instance, it is emphasized that the organism responsible for Malta fever is barely distinguishable from the *Brucella abortus* which causes the infectious abortion of cattle. Quite recently in America persistent pyrexia in human beings has been caused by the same *Brucella abortus* ingested in cow's milk.

The authors have given space to the Salmonella group and make mention of the *Bacillus psittacosis*. In view of recent reports a further discussion of the relation between the well defined enteritis from which parrots suffer, and the vague, atypical pneumonia which in the human is called "psittacosis," would have been welcome.

The nomenclature adopted is that recommended by the Committee of the American Society of Bacteriologists in 1920. Thus the organism responsible for tuberculosis is referred to as the "*Mycobacterium tuberculosis*." The bacillus of diphtheria has become the "*Corynebacterium diphtheriæ*"; that of plague the "*Pasteurella pestis*." Fortunately in the text the authors descend to the names with which we are more familiar.

The plates are excellent and the references are complete.

[1] "The Principles of Bacteriology and Immunity," by W. W. C. Topley, M.A., M.D., M.Sc., F.R.C.P., and G. S. Wilson, M.D., M.R.C.P., D.P.H.; Volumes I and II; 1929. London: Edward Arnold and Company. Royal 8vo., pp. 1231, with illustrations.

ECLAMPSIA.

THE first English edition of "The Improved Prophylactic Method in the Treatment of Eclampsia," by Professor W. Stroganoff, will be welcomed by all who wish to have a first hand knowledge of Professor Stroganoff's methods.[1]

A review of various methods which have been adopted in the treatment of eclampsia, is given and then the history of the prophylactic method is traced. The object of the prophylactic method is to interrupt the fits by acting on the patient with all the means and measures at our disposal. The measures discussed are:

1. Removal of all possible irritations.

2. The introduction of narcotics. The chief narcotics used are chloroform, morphine given subcutaneously, chloral hydrate given by mouth or by the rectum.

3. Delivery. The promptest possible is advocated, but not forcible delivery. Cæsarean section is regarded as a contributory factor towards a fatal issue and in patients who recover, a means of unnecessarily deforming a woman.

4. The upkeep of the main functions of the body. The value of oxygen subsequent to each fit until asphyxia completely disappears is stressed.

5. Venesection. The indications are given for its use and good results are claimed when it is used in conjunction with other means and the amount of blood removed is comparatively inconsiderable.

The main features in which this method differs from all those previously suggested, consist in: (i) A systematic and regular introduction of narcotics as a preventive against fits, (ii) the application of various narcotics, (iii) the introduction of narcotics until all definite signs of eclampsia disappear. The fits must be interrupted and if they recur, narcotics must be introduced in larger doses and even more frequently, while in the interval between the application of the essential narcotics chloroform narcosis should be more freely administered. Chloroform in small amounts is held to be preferable and safer than ether.

The book can be recommended as a valuable contribution to the literature on eclampsia and shows again what excellent results can be obtained by conservative methods as opposed to surgical procedures.

[1] "The Improved Prophylactic Method in the Treatment of Eclampsia," by Professor W. Stroganoff; Third Edition, thoroughly revised and completed; First English Edition; 1930. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 160. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, MAY 31, 1930.

The Health of the Pacific.

IN another place in this issue there appears a *résumé* of a report by Dr. P. Hermant and Dr. R. W. Cilento on health conditions in the Pacific Islands, recently issued by the League of Nations Health Organization, Geneva. This report should be studied with care. It deals with matters of vital and national importance. The Commonwealth has responsibilities in the Pacific which it cannot shirk. Even if the rank and file be insensible to their duty to other peoples, they may surely be pervailed upon to consider the subject from the point of view of their own national health. If the health of Australia is to be conserved, she must be surrounded by healthy nations.

The problems of the Pacific first received serious consideration at the Pan-Pacific Science Congress held in Melbourne and Sydney in 1923. The proceedings of the Section of Hygiene were fully reported in this journal at the time and included important papers on the depopulation of the Pacific by Dr. J. H. L. Cumpston and Dr. R. W. Cilento. As time went on it was recognized that the matter could not be allowed to rest and as a result of conversations between the British Colonial Office, the United States Government, the French Colonial Office and the Commonwealth Government it was decided that an international Pacific Health Conference should be held in Australia. This decision had the cordial approval and support of the League of Nations—the League was represented at the conference by Dr. Norman White. It is to be noted in passing that this conference, held in 1926, remains the only international health conference with accredited diplomatic representatives that has been held in Australia. The members of the conference recognized the importance of their deliberations and adopted a wide outlook. They discussed such questions as quarantine agreements, rapid dissemination of information based from Australia and further investigations by concerted action between the parties concerned. A recommendation was passed to the effect that the League of Nations Health Organization, including the committee of *L'Office International d'Hygiène Publique,* be invited to give practical assistance to epidemiological and preventive work in the Austral-Pacific zone. It was suggested that three possibilities should be considered: (i) The initiation or promotion of schemes of coordinated research in the Austral-Pacific zone; (ii) the taking of such steps as were necessary to consult the governments concerned as to the practical form of such schemes; (iii) the securing of the cooperation and assistance of other international health organizations for this purpose. The Commonwealth Government was invited to take such action as it considered appropriate to bring these considerations to the notice of the administrations and international organizations concerned. After this recommendation had received the approval of the Assembly of the League of Nations, the matter came before the Health Committee of the League. Dr. C. L. Park, Chief Medical Officer on the staff of the High Commissioner for Australia in London, was invited to make a statement in regard to what action might be possible to give effect to the resolution, more particularly in regard to a preliminary and limited local inquiry. Sir George Buchanan, the delegate for Great Britain to the International Pacific Health Conference, endorsed the proposal that the inquiry should be limited to an exploratory mission by one or two people to decide what were the points on which investigation would be of the most value for the islands in question and also for the advancement of epidemiological knowledge. The Health Committee of the League of Nations, at its eleventh session in 1927, expressed the opinion that a survey of the health conditions in Papua, New Guinea, the New Hebrides, New Caledonia, the Solomon Islands and Fiji would yield results of international interest and importance. The Director-General of the Commonwealth Department of Health was requested to study the possibilities

of making the necessary arrangements for carrying out such a survey at an early date. After discussion the mission was appointed. The French expert was Dr. P. Hermant, *Médecin Principal de l'Assistance Médicale de l'Indo-Chine,* and Dr. R. W. Cilento, Director, Division of Tropical Hygiene, Commonwealth Department of Health, was chosen as the Australian expert. Dr. Hermant and Dr. Cilento left Sydney on October 27, 1928, to undertake the first portion of the investigation. They returned to Sydney on April 6, 1929. The report of their survey is that which is summarized in this issue.

It will be noted that Dr. Hermant and Dr. Cilento group the problems of Melanesia under three headings: The investigation and control of specific diseases, the education of the native in medicine and the question of the survival and increase of the native races. These are practically identical with those considered at the congress of 1923 and the conference of 1926. The problems are recognized, the need is definite. Results will be obtained by cooperation and the cooperation must be along the lines of scientific intercommunication rather than in any attempt at uniformity of administration. Scientific intercommunication calls for a clearing house for information. This in virtue of her position, Australia must provide and herein lies her responsibility. Not only must she act as a clearing house, but she must provide the necessary scientific inspiration and must be prepared, if need be, to assume leadership. There is one factor that will help her and that is ready to be called into use. In 1926 all parties to the International Health Conference understood that it was necessary to reconstitute the Australian Institute of Tropical Medicine, situated at Townsville, and to provide facilities for teaching public health and tropical medicine on modern lines at a University school at Sydney. The merging of the Australian Institute of Tropical Medicine into the School of Public Health and Tropical Medicine at the University of Sydney has already been traced in these pages. It was pointed out that the new school is a school for the whole of Australia and beyond. It should be used as the central point of cooperation in the Pacific.

Current Comment.

MIKULICZ'S DISEASE.

IN 1888 Mikulicz described before a German Medical Society a case of chronic symmetrical lymphomata of the lachrymal and salivary glands, a disease to which his name has since been applied. This disease begins in the lachrymal glands and may be confined to them; but it usually extends. The swelling of the glands is hard and apparently not inflammatory. The lymphatic glands are not affected. There are no blood changes and no deviation from general health is found. No cause was discovered. Mikulicz believed the condition to be due to an infection. Examination of a removed lachrymal gland disclosed widespread round celled lymphocytic infiltration of the sustentacular tissue, as in lymphadenoma. Mikulicz stated that there was no disturbance of the salivary or lachrymal secretion.

The main features, as summarized by Robert Hutchison in 1911, are dense brawny swellings of the glands mentioned which may be tender on pressure, but are not painful. They are freely movable under the skin, but may be adherent to subjacent tissues. The facial appearance is typical. The cheeks are conspicuously broadened, as in mumps. The eyelids on the temporal side are drooped like those of a bloodhound. Elevation of the lid at the external canthus may reveal downward displacement of the retrotarsal fold by the pendulous swollen lachrymal gland. The parotid regions are occupied by broad oval tumours which may be knobby or lobulated and which may displace the lobes of the ears upwards and outwards. The submaxillary salivary glands may be prominent on the floor of the mouth, but as a rule do not make the skin surface protrude. Hutchison states that there may be dryness of the conjunctiva and mouth, difficulty in swallowing and respiratory disturbances.

Thursfield in 1914 stated that there may be in addition to Mikulicz's disease proper, a similar affection with lymphatic involvement and that lymphatic leuchæmia, tuberculosis, syphilis and gout may exhibit similar bilateral swellings of the salivary glands. Further that there is a similar congenital hereditary or family affection. He describes an intermittent periodic swelling under the name *Sialodochitis fibrinosa.* Delzet in 1914 divided swellings with the Mikulicz syndrome into either simple lymphadenoid enlargement, lymphomatose infiltration, chronic inflammatory swelling and possibly tuberculous or syphilitic enlargement.

J. P. Crozer Griffith has given a summary of our knowledge to date.[1] He has also investigated allied affections of the salivary and lachrymal glands, similar clinically, but of different ætiology. The term Mikulicz's syndrome may be used to include all disease conditions exhibiting the peculiar involvement of the salivary and lachrymal glands. As the condition is uncommon, Crozer Griffith reports

[1] *American Journal of the Medical Sciences,* December, 1929.

fully two additional cases. One is apparently an example of Mikulicz's disease proper; it occurred in a mulatto female, aged nine years. The other was a case of leuchæmia bearing clinical resemblance to Mikulicz's disease. Griffith holds it unreasonable to base a sharp distinction on the presence or absence of moderate enlargement of some lymphatic glands, because the morbid process in the salivary gland may originate in the lymphoid nodules normally present and implication of lymphatic tissue elsewhere might be expected. He also states that moderate enlargement of lymphatic glands in various parts of the body is so common that its association with the salivary and lachrymal disorder may be only fortuitous. His contention on this point cannot be denied. It is within the experience of all medical practitioners that small indurated glands may be found in the neck or groin resulting from some previous infection which may have been forgotten by the patient. Howard has stated that in a series of cases links are shown from one form of salivary gland disorder to more widespread diseases, such as leuchæmia. Others have rightly disputed this claim. In Mikulicz's disease there is a lack of uniformity in the histological findings. The round-celled infiltration of the tissues in the benign cases of Mikulicz's disease proper may resemble that found in rapidly fatal leuchæmia. Besides Mikulicz's disease proper and its rare congenital familial form, Schaffer and Jacobson have stated that the Mikulicz syndrome may be simulated by leuchæmia, tuberculosis, lymphosarcoma, gout and lead and iodide intoxication and possibly syphilis. They also mention a condition termed *Febris uveoparotidea subchronica*, described by Heerfordt in 1909. In this disorder iridocyclitis is the prominent feature, associated with enlargement of the parotid and often other salivary glands and lymphatic glands also. Its relationships are quite obscure.

In considering the cause of Mikulicz's disease, it is necessary to distinguish sharply between Mikulicz's disease proper and conditions presenting the Mikulicz syndrome. Failure on the part of many writers to do this has led to confusion. The cause of Mikulicz's disease proper is unknown. Although Thursfield suggested that it is due to inflammatory or irritative processes in the pharynx, mouth or conjunctiva, there is no evidence of the nature of the infecting agent. Perhaps an organism, as yet unknown, may be the causal factor. It is difficult to conceive any of the more common organisms producing this rare syndrome unless, in addition, some other and at present unknown factor be postulated. When association with tuberculosis, syphilis or streptococcal infections elsewhere is proved, the organisms of these conditions will possibly be responsible. Sarcoma may affect both salivary and lymphatic glands. The influence of syphilis is not certain. Some patients presenting the syndrome have improved under antisyphilitic treatment, but there was no definite evidence of syphilis being present.

The main interest centres about the relationship of this disorder to tuberculosis. Histological examination of the lachrymal gland of Crozer Griffith's patient revealed a process typical of tuberculosis, except that the presence of more necrotic material and probably less fibrosis would have been expected. However, no tubercle bacilli could be found and tuberculosis could not be produced in guinea-pigs by subcutaneous inoculation with glandular tissue. The Wassermann test on the patient gave no reaction. Examination of the urine and the cutaneous and intracutaneous tests for tuberculosis were also without result. The question at once arises as to whether, in the absence of tubercle bacilli, the histological changes were actually tuberculous or whether other causes could produce appearances previously considered to be due to the tubercle bacillus. Igersheimer and Pöllot have concluded that in Mikulicz's disease the tubercle bacillus may cause the condition, but that as a rule it is not responsible. They also conclude that the presence of epithelioid tubercles and giant cells in the gland is not absolute proof of the existence of tuberculosis. Baumgarten admitted the close similarity of the histological appearance to that of tuberculosis, but insisted that positive diagnosis could be established only by results of animal inoculation or the finding of the tubercle bacillus. The reports of many authors might be quoted. Some observers describe the apparent giant cells as "pseudo-giant cells." There is rarely any evidence of caseation and the hardness of the gland, the tendency to connective tissue overgrowth is not in accord with the typical picture of tuberculosis. One is forced to the conclusion that a histological appearance suggestive of tuberculosis is not positive proof of that disease and while the tubercle bacillus may sometimes cause this syndrome, it is an unusual cause and the explanation must be sought elsewhere. The primary change in the glands is an extensive deposit of small round cells which compress or displace the glandular parenchyma. The cells may be an infiltration of leucocytes or a proliferation of the nests of lymphadenoid tissue present in the glands. These changes, however, are not uniformly or solely seen. One possible suggestion is that the disease is a new growth—a benign neoplasm. Further speculation, however, until more data are available is futile. The hardness of the glands and tendency to connective tissue overgrowth may suggest a chronic inflammatory process.

A HOME FOR PATIENTS WITH CANCER.

THE Secretary of the New South Wales Homes for Incurables at Ryde has asked us to announce that beds are usually available for patients in the Cancer Home. This home is distinct from that to which patients suffering from other incurable conditions are admitted; for the latter there is always a long waiting list. The Cancer Home is waiting to be used.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Gastric Motility, Anhydræmia and Vitamin B.

W. B. ROSE, C. J. STUCKEY AND G. R. COWGILL (*American Journal of Physiology*, February, 1930) have shown that in vitamin *B* deficient dogs there are profound disturbances of the motor function of the stomach, even gastric atony, long before neuromuscular symptoms develop. The constancy with which normal hunger contractions were reestablished by vitamin *B* therapy, suggested the possibility that the causative factor involved physico-chemical disturbances of the blood rather than actual morbid changes in the stomach itself. By vitamin *B* here is meant the combination of factors which have been shown to make up this substance. Since anhydræmia has been shown to occur in animals with vitamin *B* deficiency, it was sought to determine whether the gastric effects might be related to this anhydræmia. Anhydræmia was produced in normal dogs by giving them only the same amount of water as taken by the vitamin *B* deficient dogs. The anhydræmia that ensued, was associated with complete gastric atony in all cases, with a rapid return of hunger contractions when water was administered. It appears, then, that the decrease in gastric motility occurring in vitamin *B* deficiency is due in part at least to the concomitant anhydræmia which develops.

Permanent Results of Denervation of a Cutaneous Area.

THREE years ago E. Sharpey-Schafer (*Quarterly Journal of Experimental Physiology*, February, 1930) had the branch of the ulnar nerve distributed to the radial side of the little finger of his left hand cut. He has studied the permanent effects of this section, sufficient time having elapsed for complete regeneration to have taken place. The sensation of touch is abolished or at any rate obscured by a concomitant sensation of pain. Appreciation of warmth and cold has returned, but in a somewhat diminished degree as compared with a normal area. Sensation of pain is increased over the originally denervated area as compared with that felt over a normal area. Trophic conditions are less favourable than in parts where there has been no interference with the nerve supply.

Influence of Diathermy on Gastric Secretion.

C. E. STEWART AND W. N. BOLDYREFF (*Bulletin of the Battle Creek Sanitarium*, October, 1929) have studied the effects of diathermy on the gastric secretion and gastric motility of sixteen dogs with gastric fistula, gastric and œsophageal fistulæ and isolated gastric pouches. In the state of rest of the gastric glands (with an empty stomach and absence of secretion of gastric juice) diathermy stimulates the gastric secretion. Normal results were usually obtained in pathological conditions of the stomach. As far as the properties of gastric juice are concerned, after diathermy higher acidity was observed and apparently lower content of pepsin. Diathermy is capable of increasing secretion of gastric juice in the psychical phase of gastric secretion, as well as in the chemical phase. The properties of gastric juice, secreted in the psychical and chemical phases of digestion, are altered by the action of diathermy in the same way as in the case of an empty stomach. Diathermy has an inhibitory effect upon the contractions of the stomach during the absence of gastric secretion. Diathermy also stimulates the secretion of pyloric juice.

Male Hormone.

MANY attempts have been made to isolate a male hormone, but these have ended in failure, mainly because no adequate tests were employed to measure the potency of the extracts. The castrated cock can be used as a test animal, the growth of the comb and wattles being taken as a measure. As the male hormone has been shown to be present in the blood by Pezard, it seemed logical to conclude that it would probably be also found in the urine. C. Funk, B. Harrow and A. Lejwa (*American Journal of Physiology*, March, 1930) have obtained extracts from the urine of young men which when injected into capons induced comb growth. The extracts prepared from testicles, when injected, also gave definite but less pronounced effects. The difference may depend on the amount of material used. Administration *per os* also had some effect, but the effect was much less pronounced and less constant than when the extract was injected. The male and female hormones showed marked similarity in chemical properties. Both may be extracted with chloroform, both form alkaline salts, both are found in the fatty acid fraction and the methods of purification so far adopted apply to both hormones.

Passage of Inert Materials Through the Intestinal Tract.

VARIOUS investigators have used glass beads as a test material to determine the rate of progress of food residues through the bowel. F. Hoelzel (*American Journal of Physiology*, March, 1930) has examined a number of inert substances as well as beads to determine whether there was any difference in the time of passage. The test materials included rubber, cotton thread knots, seeds, glass beads, aluminium, steel, silver and gold and the animals used were rabbits, guineapigs, dogs, cats, rats, mice, monkeys, pigeons, hens and man. Rates of passage more or less proportional to the specific gravity of the test materials were found, the heavier materials passing slower than the lighter materials. The rates of passage also varied considerably in different species of animals. The main sites of stasis were determined in some of the animals. In rabbits, for example, the stasis occurred most regularly in the duodenum and to a lesser extent in the colon, caecum and stomach. In man heavy materials tended to accumulate mainly in the caecum, colon and terminal portion of the ileum. In man glass beads showed an average rate of passage of 40·15 hours while recognizable food residues took 25·0 hours and steel balls 79·98 hours. Heavy test materials tend to reveal changes in motility more readily than light test materials. Silver would probably be heavy enough to reveal any serious alterations in man. A light test material opaque to X rays, such as small hollow balls of silver sealed or filled with paraffin, might be useful in trying to locate local sites in the digestive tract with abnormal stasis or segments with partial obstruction where a powder like barium sulphate would pass too easily or might be washed through.

"Amytal" and the Inhibitory Action of the Vagus on the Heart.

"AMYTAL," isoamyl-ethyl-barbituric acid, has found favour in recent times as a non-volatile anæsthetic, especially for laboratory animals. R. C. Garry (*Journal of Physiology*, March, 1930) has found that after cats have been anæsthetized with "Amytal" stimulation of the peripheral ends of the right or left peripheral vagus failed to give the expected result on the heart. A number of experiments have been carried out on cats and rabbits with "Amytal" anæsthesia. In all cases the inhibitory action of the vagus was either completely absent or extremely slight with a strength and duration of stimulus capable of giving a well marked action before the administration of "Amytal." The effective anæsthetic dose is given as fifty milligrammes per kilogram body weight. Given intravenously, even less than fifty milligrammes per kilogram partially or totally obliterated the action of the vagus.

BIOLOGICAL CHEMISTRY.

The Effect of the Diet on the Iron and Copper Content of the Hen's Egg.

C. A. ELVEHJEM, A. R. KEMMERER, E. B. HART AND J. H. HALPIN (*Journal of Biological Chemistry*, December, 1929) have investigated the iron present in the egg and the chick and have found that the margin between the amount present in the egg and the quantity needed to produce the hæmoglobin in the chick is very small. The total iron content of an egg of average size is about 0·8 to 1·0 milligramme and the iron content of a day-old chick determined by actual analysis or by calculation of the amount in the hæmoglobin of a newly-born is between 0·6 and 0·7 milligramme. Thus very little iron can be wasted during the development of the egg into the chick and the chick has no source of iron from which additional hæmoglobin can be built. The work that has been done on the effect of diet on the iron content of the egg, is of a conflicting nature and

it is difficult to decide which data are correct. Since copper and iron are so closely related in hæmoglobin building, these workers investigated the effect of the hen's ration on the iron and copper content of the egg yolk and the copper content of the egg white. Experimental methods and the methods of analysis are given in detail. Figures obtained show egg albumin to be practically free from iron, but to contain almost as much copper as the yolk. The figures also show that the copper content of the yolk and white is not influenced by feeding either iron alone or iron together with copper to the levels used in this investigation. The quantity of iron in the yolk could not be increased by feeding either iron alone or iron and copper.

Epinephrine and Blood Sugar Concentration.

C. F. CORI AND G. T. CORI (*Journal of Biological Chemistry*, November, 1929) have performed experiments on seven medical students to discover whether subcutaneous injection of epinephrine causing hyperglycæmia is accompanied by a rise in the arterio-venous blood sugar difference. The subjects came to the laboratory without breakfast and after a period of rest blood was taken from the radial artery and from the cubital vein without stasis. One cubic centimetre of one in one thousand adrenalin solution was injected subcutaneously. Observations of pulse rate and blood pressure were taken at frequent intervals and one hour after the injection a second sample of blood was taken. No rise in the arterio-venous blood sugar difference was found. It was concluded that the excess of sugar accumulating in the blood after epinephrine injections does not evoke the same response as alimentary hyperglycæmia.

The Acid Base Equilibrium of the Blood in Eclampsia.

H. J. STANDER, N. J. EASTMAN, E. P. H. HARRISON, JUNIOR, AND J. F. CADDEN (*Journal of Biological Chemistry*, December, 1929) have undertaken a study of the acid-base equilibrium of the blood in eclampsia. Investigations were made into the chemistry of the blood in normal non-pregnant women, normal pregnant women and in eclamptic women. The following constituents were investigated: The hydrogen ion concentration of the blood, carbon dioxide content, total bases, chlorides, proteins, non-protein nitrogen, acetone bodies and organic acids and also the organic acids in urine. The methods used are given and also full details of the way in which the specimens of blood were collected. Normal pregnancy was found to be associated with a reduction in total serum base accompanied by a decrease in serum protein and serum bicarbonate. The other anion constituents were within the normal limits associated with the non-pregnant state. No increase in organic acids or accumulation of abnormal acids was found during normal pregnancy. In uncomplicated gestation the hydrogen ion concentration of the blood was found to be within normal range. All the eclamptic patients studied had a true acidosis due to an uncompensated alkali deficit with a definite accumulation of organic acids (probably chiefly lactic) in the blood. So far normal values for blood acetone bodies have been found and ketosis probably does not play a part in the production of this acidosis. The organic acids in the urine in eclampsia were as a rule greatly increased. The hydrogen ion concentration of the blood in eclampsia was found to be definitely lowered, averaging about 7·04. From these results it is evident that the uncompensated acidosis in eclampsia is so pronounced that it in itself may cause the death of the patient. Whether the eclamptic convulsions cause this low pH value or are the result of it has not so far been determined. The authors conclude that this acidosis deserves special attention in treatment and intensive antacidosis treatment is advocated.

Creatine-Creatinine Metabolism.

DURING the course of some studies on the physiology of the liver with reference to creatine-creatinine metabolism, Jessie L. Bollman (*Journal of Biological Chemistry*, December, 1929) found several dogs in which continued administration of creatine failed to give rise to an increase in the urinary excretion of creatinine, observations which did not correspond to well established experiments of others. The creatine was administered intravenously and not orally and the protein content of the creatinine free diet was lower than that used by other observers. Well nourished male dogs were used for these experiments. This worker found that with a creatine-free diet low in protein, excretion of creatinine was not altered by daily feeding or by daily intravenous administration of creatine in amounts comparable to the daily excretion of creatinine. Considerable amounts of the administered creatine were recovered unchanged in the urine. The addition of large amounts of protein to the diet greatly reduced the amount of creatine recovered in the urine. After two weeks of protein feeding and administration of creatine the excretion of creatinine began to increase and in some cases remained increased for a few weeks after the high protein diet was discontinued. This seemed to the author to be direct evidence of the conversion of creatine to creatinine and to be also indicative of the relation of this conversion to the existing level of the metabolism of protein.

The Influence of Prolonged Muscular Rest on Metabolism.

DAVID PATON CUTHERBERTSON (*Biochemical Journal*, Volume XXIII, page 1328) has investigated the metabolic changes produced by prolonged muscular rest. The experiments carried out were concerned with pure disuse atrophy and not with the atrophy due to destructive lesions of the lower motor neurones or their axones or to toxic action or to pressure. The subjects of the experiments were either volunteers in perfect health or patients admitted to hospital with loose fragments of cartilage in the knee joint who suffered no discomfort and were in good health. The food and water intake was kept constant throughout and a preliminary period of four or five days was allowed for the subject to obtain nitrogenous equilibrium after which the subjects were confined to bed. Within a day or two from the commencement of a period of muscular rest the subjects showed a rise in the excretion of sulphur, nitrogen, phosphorus and calcium in that order. This loss was maintained fairly steadily for a varying period after which it gradually declined. The rise in the output of sulphur was due to a slightly greater proportionate excretion of inorganic sulphates. There was a slight fall in the excretion of ethereal sulphates, the neutral sulphates remaining more constant. The rise in the excretion of nitrogen was mainly due to a proportionate increase in the amount of urea. The excretion of ammonia, following an initial fall at the commencement of the rest period, took longer to rise, but continued parallel to the total nitrogen. The excretion of fæcal nitrogen remained practically constant. The excretion of creatinine and uric acid remained practically unaltered. The sulphur-nitrogen ratio obtained suggested a sulphur rich source of the excreted material, presumably for the most part muscles. The data provided from these experiments as to the part played by calcium and phosphorus were inconclusive and longer periods of immobilization are necessary with longer preliminary periods before satisfactory figures can be obtained. Apart from a slight fall in the oxygen consumption associated with a rise in the respiratory quotient, the gaseous metabolism remained very constant from day to day.

Cerebro-Spinal Fluid Calcium.

IT has been suggested that the cerebro-spinal fluid calcium might be used as a measure of the diffusible calcium of the blood plasma. D. M. GREENBERG (*Proceedings of the Society of Experimental Biology and Medicine*, March, 1930) has investigated this possibility. It is found that when the blood calcium is at a normal stable level, there is an approximate approach to an equilibrium between blood and spinal fluid and the spinal fluid calcium is then a close measure of the diffusible calcium. When the blood constituents are undergoing definite fluctuations, the spinal fluid changes do not keep pace. The hypothesis of a near approach to equilibrium in cases of a normal stable level of the blood constituents and the failure to keep pace with marked fluctuations also harmonize with the approximate agreement to a Donnan membrane distribution found in some instances and a wide departure in others noted by Hamilton.

Special Abstract.

HEALTH CONDITIONS IN THE PACIFIC ISLANDS.

DR. P. HERMANT AND DR. R. W. CILENTO who were entrusted with a survey of the health conditions of the Pacific Islands, 1928, have made their report.[1]

In discussing the administrative subdivision of Melanesia, the authors of the report point out that Melanesia comprises New Guinea with the Bismarck or New Britain Archipelago, the British Solomon Islands, the New Hebrides, New Caledonia and its dependencies and Fiji together with some thousands of small outlying islands. With the exception of Fiji which may be regarded as the junction point between Melanesia and Polynesia, this vast island chain follows a course parallel to the north-eastern coast of Australia. To the north Melanesia is bounded by the island of Micronesia, to the west by Malaysia and to the east by Polynesia. Racial intrusions from each of these areas have modified the composite Papuo-Melanesian stock that peoples Melanesia. Though there are variations in skin colour, the texture of the hair and general physical characters, the population is essentially identical from New Guinea to Fiji. Uniform in race, but modified by intruding racial factors, the Papuo-Melanesians are uniform also in disease conditions and problems throughout 90% of Melanesia, but in the 10% remaining the most recent intruding racial strains colour the picture. Along with these elements of unity, however, the natives of this area are abruptly dissociated by a multitude of dialects and by the fact that their total territory is subdivided into six separate administrative zones. The health conditions and problems of Melanesia are described under two headings, those of north Melanesia and those of south Melanesia. North Melanesia comprises the territory of Papua (formerly British New Guinea), the territory of New Guinea (formerly German New Guinea), administered by Australia under mandate, and the British Solomon Islands Protectorate. South Melanesia includes the colony of Fiji, New Caledonia and its dependencies and the *Condominium* of the New Hebrides.

A description of each of these territories is given together with a short historical account of the forms of administration which have been adopted. An account is also given of the existing medical services and of the problems encountered by each. It is not necessary to consider these *seriatim*; they receive consideration in the general discussion.

It is concluded that the public health picture of Melanesia presents several distinct aspects. The vital question of the distribution of malaria comes first. This divides the group into two areas. One is free from malaria and comprises Fiji and New Caledonia with its dependencies. The other is heavily infected and includes the territory of Papua, the Mandated Territory of New Guinea, the Protectorate of the British Solomon Islands and the *Condominium* of the New Hebrides. The second great factor of importance is that of Asiatic immigration and this has affected to a great extent the disease picture in areas which are otherwise similar. The distribution of amœbic dysentery appears to coincide almost exactly with the distribution of Asiatic population. The same holds true in regard to the distribution of ankylostomiasis, ascaridiasis and probably other parisitic diseases. Fiji has a large population of Indians, almost equivalent in numbers to the native Fijian population. The Indians have certain definite disease characteristics which have tended to obscure and to modify the disease picture of the indigenous Melanesian. In New Caledonia a similar condition exists, but here it tends to become even more pronounced because, if the Loyalty Islands which have no Asiatic immigration are excluded, the indigenous population in that locality amounts to only 17,000. During the last five years Indo-Chinese labour has been introduced into the New Hebrides. A total of some 5,000 persons has been introduced and this number is increasing. Problems similar to those existing in New Caledonia and Fiji will presumably arise. In Fiji, where the Indian population is now fixed, immigration is small and indenture has ceased; the process of modification may be said to be relatively at an end. The Chief Medical Officer of Fiji who adds an appendix, points out that the introduced Indian population is as much influenced by its Melanesian surroundings as it previously influenced the Melanesian disease picture. Throughout the whole of Melanesia under observation there are three almost universally prevalent specific diseases, filariasis, frambœsia (yaws) and hookworm infestation.

In regard to the economic question, the most vital is the possibility of survival or increase of the native population. Sociological factors have a profound influence in this regard. The authors of the report point out that observers have devoted attention to various aspects of this matter and have attached several degrees of importance to questions of psychology, to matters of diet and so forth. In addition to the three diseases already mentioned there are others which have to be taken into consideration. These include leprosy which is prevalent to an enormous extent in south Melanesia and to an undetermined extent in north Melanesia, tuberculosis and other respiratory infections which are relatively widespread in the neighbourhood of white settlements wherever investigation has been thoroughly made, bacillary dysentery which now appears to be mildily endemic in most parts, gonorrhœa which is prevalent only in areas where labourers have returned from indenture, but which is of a particular type.

It is stated that the problems of Melanesia can in general terms be grouped under three headings: (*a*) The investigation and control of specific diseases, (*b*) the education of the native in medicine and (*c*) the question of the survival and increase of the native races.

Specific Diseases.

Frambœsia.

Frambœsia is almost universal in Melanesia in the coastal areas and extends inland to a varying distance. The disease responds so readily to organic arsenic preparations that the use of these drugs has become an important factor in extending the influence of medicine to suspicious or hostile tribes. The absence of syphilis among natives and the relationship between frambœsia and syphilis are important considerations in south Melanesia on account of Asiatic immigration. This question is not so important in north Melanesia where there is no record of the occurrence of syphilis in a native. The question is raised as to whether mass treatment of frambœsia will leave the field open for the introduction of syphilis and it is pointed out that the cost of organic arsenic preparations is of practical importance.

Hookworm Disease.

The indigenous hookworm throughout the whole of Melanesia is *Necator americanus*. It is extremely widespread, but appears to be of little significance or importance in actual hospital statistics. It probably acts by reducing the resistance to other diseases and in limiting powers of recuperation. In most areas it is possible to cope with the situation by occasional mass treatment. These conditions do not obtain when the causative organism is *Ankylostoma duodenale*. This organism is associated with Asiatic immigration. Its virulence is much more pronounced than that of necator, it is much harder to expel and it is not affected to the same degree as necator by carbon tetrachloride. It is thought that the ankylostome is a factor of importance in certain European communities throughout the Archipelago. The view is expressed that the measures of control adopted in Queensland would yield satisfactory results in such localities. These consist in the examination of school children, treatment of infected persons, "follow-up" work by treatment of parents and other residents and adequate sanitation of the home. In these circumstances it would be necessary to include ankylostomiasis among the notifiable diseases.

[1] Report of the Mission Entrusted with a Survey on Health Conditions in the Pacific Islands, by Dr. P. Hermant and Dr. R. W. Cilento; League of Nations, Health Organization, Geneva, 1929.

Filariasis.

Filariasis is almost universal in Melanesia. It is a potent factor in economic loss. Elephantiasis, apart from the disfigurement which it causes, cripples a definite proportion of the natives and may have a minor effect on population. Among the manifestations of filariasis are abscesses of muscle; these may be either filarial or non-filarial in type. It is only recently that attention has been devoted to myositis in the Pacific as to a specific entity unrelated to filariasis. The percentage of persons affected varies greatly from place to place—100% to complete absence. Sayers has found areas which are free from filariasis or elephantiasis and it is noticed that in such areas myositis exists as commonly and occasionally more commonly than elsewhere where filariasis is rife. It is suggested that this demands investigation. Other problems requiring investigation are the question of periodicity and that of the vector of the disease which has been actually established only in some few localities.

Malaria.

As already stated, the presence of malaria serves to distinguish north from south Melanesia. The possibility of its introduction into Fiji or New Caledonia calls for serious consideration. The factors which prevent the introduction of the disease have not been definitely determined. It is considered that malaria does not exist in either territory because anophelines have not been found. There is, however, no adequate evidence of the non-existence of anophelines other than the accepted fact of the absence of malaria and the fact that anophelines have not been discovered by any entomological collector. It is considered most desirable that there should be a comprehensive entomological survey of the whole of Melanesia in regard to the question of the vector or vectors of both filariasis and malaria. Stress is also laid on the necessity for an examination of the whole of the literature dealing with the mosquito fauna in Melanesia. This is looked upon as one of the most important features of coordinated research which could be instituted at the present time. Since most of the administrations have neither the men nor the money for such an undertaking, it would be necessary for the scheme to be financed from outside.

Venereal Diseases.

As already pointed out, syphilis practically does not exist except as a possibility of the future. On the other hand gonorrhœa is found to be fairly common. The type of the infection is unusual in that it produces very few complications and the question has been raised in some localities as to whether the disease is actually gonorrhœa or not. As intercourse between the tribal groups becomes more frequent and intermarriage more usual, it is probable that the disease will progress.

Dysentery.

Bacillary dysentery is not the scourge that it formerly was and the provision of adequate serum treatment aborts it with dramatic suddenness. Amœbic dysentery, on the other hand, demands careful consideration wherever it is found. Since this question is one of local routine, it is not considered that profitable coordinated research could be recommended.

Tuberculosis and Other Respiratory Diseases.

In certain of the administrations in Melanesia tuberculosis is regarded as the most important feature in the problem of depopulation. The findings of the Mission confirm the view expressed by Buxton in his report on the New Hebrides, that there is some factor which outruns European penetration or civilization and which has a deleterious effect on the natives and that this factor is probably tuberculosis. As a result of the examination of the bodies of native labourers dying at Rabaul it has been found that 24% of all indentured labourers die of tuberculosis and that pneumococcal infection accounts for an equivalent number. In other words half the total deaths of labourers are due to pneumonia or tuberculosis. Investigation shows that the native develops but little resistance to diseases of this nature. It is suggested that he has not acquired the partial immunity found among European peoples. At the same time there is another factor to be considered—the food supply. The surprisingly good effects in certain localities of cod liver oil treatment and the important observations in relation to dietetic deficiencies indicate that in this field there may be an important factor in regard to survival.

Food Deficiencies.

The question of food deficiencies is one of the greatest factors in the health picture of Melanesia. Even at its best the diet of the Melanesian in his own village is bulky, innutritious, difficult to digest, deficient in fat and in protein and often poor in vitamins *A* and *C*. The deficiencies of the diet are made up by the use of the growing shoots of plants, certain grasses, ferns and fruits, the raw liver of certain animals and various kinds of fish. Both liver and fish of the type rich in vitamin content are regarded by natives as of the utmost value. They were used as the stable protein food of the fighting population. It is pointed out that in the Solomons, for example, the whole adult life of the male native was devoted to shark fishing and man-hunting and also to pig raising. Where the native is visited only rarely by white men, he continues to devote his energy to the obtaining of his traditional foods and flourishes. When he comes into contact with Europeans, cannibalism is forbidden and both pig raising and the hunting of large fish rich in vitamins are greatly diminished. This is associated either coincidentally or as an actual sequel with a very great loss of population, a loss which continues and is increased by such factors as alienation of land, recruitment of the most virile portion of the community and so forth. With the arrival of planters and traders and the establishment of plantations the native is pinned down to fixed localities and he is prevented from migrating from spot to spot as the fertility of the land diminishes. As a result of this and of the inelasticity of native habits he becomes resigned to an inevitable decrease in available food. Such a decrease is a well recognized factor in the diminution of indigenous population both because of the faulty nourishment of pregnant women, the diminished virility of the offspring and the increased actual failure of impregnation. There is often added a deliberate policy of abortion and infanticide—two customs of the native from time immemorial when food stocks were low. With the establishment of European plantations there often come pests which attack the food of the native in a serious manner. In the more civilized communities, such as those of Fiji, the barbaric aspects of the subject are not seen.

With the arrival of the white man there is an enormous initial decline of many native stocks, a decline resulting from many factors. Secondly, there occurs a period which is relatively stationary. During this period the native struggles to adjust himself to his altered environment and his survival picture fluctuates irregularly towards an unstable equilibrium, an equilibrium which is readily upset by any minor factor. Finally, one of two things occurs. Either the factors for decline predominate and the native race disappears or adjustment becomes gradually complete and the native population begins to express this feature in a steady increase. The first stage may be seen in the New Hebrides, the second stage in the territories of north Melanesia and the beginnings of the third stage are seen in Fiji. A strong recommendation is made that organized steps should be taken to investigate the problem throughout the whole of Melanesia and that the facts and figures recorded be collected on a basis adapted to comparison and with a view to establishing the truth or otherwise of the claim made by supporters of the food deficiency theory in depopulation.

Enteric Fever and Other Infective Diseases.

In the process of colonization of new countries the primary disease picture is coloured by malaria and subsequently as civilization proceeds enteric fever becomes the predominant feature. This happens more particularly when a non-indigenous population arrives in considerable numbers from localities in which enteric fever is endemic. This is the experience in Melanesia. At one time it was considered that enteric fever did not exist in Melanesia.

It then appeared that certain cases occurred which corresponded to the typho-malarial syndrome of earlier text books. It was ultimately observed that true enteric fever was occurring unrecognized. At the present time enteric fever has not been seen in the British Solomon Islands Protectorate. It is extremely rare in Papua. It is becoming increasingly frequent in Rabaul and is engaging the serious attention of the administration. For the New Hebrides accurate figures are not available. In Fiji and in New Caledonia it is a matter of the greatest importance and has attracted considerable administrative attention. The mission concluded that there was no point in regard to enteric fever upon which coordinated research could profitably be instituted, the disease being in every country a matter of domestic sanitation and routine hospitalization. In general the same statement applies to the commoner exanthemata.

Although measles is said to have destroyed one-third of the population of Fiji in 1874-1875 and a second epidemic years later destroyed thousands of people, it occurs at present without any virulent symptoms. The experience of Fiji has not been paralleled in any other part of the Pacific under review. In general it is concluded that measles need no longer be feared.

Diphtheria is beginning to assume the characters seen in civilized countries.

Scarlet fever does not seem to occur. It has been remarked in north Queensland, however, that occasional attacks of a disease associated with albuminuria and sore throat affect numbers of people and apparently the disease displays a certain degree of infectivity. It is thought that possibly it may be found that an organism, either identical with or similar to that causing scarlet fever, occurs in the Australasian tropics, but that the typical skin rash is obscured both amongst natives and Europeans.

Population and Depopulation.

According to statistics the population of Melanesia has fallen enormously during the last century and it is held by many that the process of depopulation is a constant one and that it represents the reaction of the native races to European contact. The members of the mission conclude that on examination at close quarters this generalization appears to be fallacious. It has already been mentioned that the effect of European intrusion upon native races may be divided into three periods. It is pointed out that practically speaking the first period has long since ended in the Pacific. The second period, that of partial adjustment, is characterized by an unstable equilibrium. This partial equilibrium is so delicate that pronounced effects may be produced upon it by circumstances which would appear otherwise to have only a casual significance. Often it is the observation of one of these factors which has caused almost every observer who has written on the question of depopulation, to regard some feature of minor importance as being the sole or greatest element in the problem. In the third period the attempted adjustment either fails or succeeds. The former is rare and is exemplified in Tasmania; the latter is exemplified in Samoa. The problem of depopulation in most instances may be solved by assisting the natives through the period of unstable equilibrium into the safeguards that insure the continuance of ordinary civilized races and, particularly from a medical view, by adjusting sociological questions. Students of the depopulation of Melanesia have suggested numerous causes. Many of these are of but slight importance. Some are drawn from local observation which is contradicted by a wider view, and some are mere adjuncts to root processes which are obscured by the more spectacular and readily observable elements. Actual destruction of native life by violence may be disregarded. Over-recruitment is now prohibited by rules and regulations. The wearing of clothes is found to have no significance one way or the other where observations have been made among populations, clothed and unclothed. Certain factors, however, are regarded as of definite importance. These factors are not confined to Melanesia, but are universal in their application and would have an effect for depopulation upon the members of any race. Whether their significance is sufficient to explain the present problem in Melanesia must be a matter for future investigation. These factors are the heavy infantile mortality rate that is observed in many parts of Melanesia, the considerable mortality rate at all ages from preventible disease and the fact that the great majority of natives live on a "border-line" diet or alternate between periods of plenty and periods of actual starvation.

The members of the mission found during their investigations that there was a paucity of authentic figures. In practically no instance was it possible to obtain data which could be analysed to provide indications of the causes affecting increase and decrease of population. It is recommended that an investigation should be undertaken as soon as possible into the question of population and that at intervals the results should be compared and contrasted so that administrative action might be planned according to the results. It is pointed out that it is insufficient to concentrate upon the provision of desirable facilities of a European nature for natives. It is not the provision of these facilities which is of importance, but their suitability, their acceptance and their best use by the natives. This statement is exemplified by experience in regard to the erection of houses of European type and the centralization of village life.

Medical Education Among Natives.

Under the caption of "Medical Education Among Natives" the members of the mission point out that the medical problems of Melanesia are complicated by many factors. Among these are the low state of civilization, the belief in sorcery and the scattered nature and the smallness of the native communities. It was suggested long ago that it would ultimately be necessary to train and employ natives in a medical service. Experiments have been made in two directions. As early as 1885 the Government of Fiji invited the chiefs to send eight or ten young men of approved intelligence and character with a view to their being given a course of instruction in the rudiments of human anatomy and physiology, medicine and surgery, together with the ultimate status after three years' service of native medical practitioner. In 1906 or 1907 a class of native women for training as midwives was instituted at a hospital under the supervision of a matron, the course being limited to six months or to assistance at a set number of deliveries. These schemes were continued for a number of years, but after a survey by Dr. V. G. Heiser, of the International Health Board of the Rockefeller Foundation, in 1916, it was thought that something more was needed if the native medical practitioner was to be a power in the country. Ultimately a Central Medical School was established at Fiji for the training of forty students. The Rockefeller Foundation agreed to contribute to this a diminishing proportion of the total expenditure for four years. After this period the responsibility for the continuance of the scheme was to fall upon the High Commissioner for the Western Pacific. Among the students were to be twenty drawn from Fiji, four from the British Solomons and several Indian students. Great things are hoped for from this scheme and it is thought that the native medical practitioners will be extremely valuable men and that it might be worth while later on extending to the best of them the privileges of full medical training at a modern university. It has been found, however, that there is an element of doubt as to the acceptance of these practitioners by the natives to whom they are sent. While in Fiji they may be accepted without reserve because of the educational advantage they have obtained, the situation will perhaps not be so fortunate in localities like the British Solomons, where the languages are many and the peoples have no common ground of interest or attachment. On account of suspicion and ignorance of the villagers the native medical practitioner has sometimes found it easier to do nothing other than carry out his duties. Close supervision by European supervisers is necessary to prevent abuse. Where the people are opposed to the scheme, native hostility is impervious even to European persuasion and the problem can be met only by prior native education.

The administration of the Territory of New Guinea has realized the difficulties of the former scheme and it has begun at the other end of the scale. An intelligent native is withdrawn from every village under control and is

trained for three months at the nearest European and native hospitals in the simple technique of the commoner native diseases. He returns to his village free from head tax, accepted as a member of the local native administration and with a title of "doctor boy" or "medical tultul." Every year he returns to the hospital for a recapitulatory course. The work done by these boys is surprisingly in excess of the very slight training they receive, but their value lies not so much in this as in the fact that they represent an intelligent service of a very widespread nature and they are an educational factor which makes civilized medicine a desirable commonplace to the natives of the village. Those now employed in the mandated territory number 2,098 and some of them have a creditable length of service. From the seniors of the native supervisers of these "boys" it is hoped to train native medical assistants who will function as effectively and over as wide an area as the white medical assistants (unqualified men with special training) who now exercise general supervision. It is thought that in some twenty years it may be possible with the betterment of education, living standards and so forth, to train native medical practitioners to fill junior posts in the medical service for natives. In the opinion of the members of the mission all these experiments will be watched with interest and it is thought that much good may be obtained by the periodical discussion of the progress made and of any modifications of policy found necessary or desirable.

Infant Welfare.

One of the largest factors in the diminution of native population is the great mortality that occurs during the first five years of life. Even in Fiji where there is no malaria, the mortality during the first year of infant life is 158·3 per thousand births. In malarious countries, such as the Mandated Territory of New Guinea, the infantile mortality varies from 120‰ to 419‰ with an average rate of 176·24‰ for all districts recorded. In this matter it is the ignorance of the village mother which is of primary importance. Probably there has always been a considerable infant mortality, but it appears that in some instances this has increased within recent years. The question of abortion and infanticide is of importance. Where polygamy was the rule, certain antisocial practices have grown up since the substitution of monogamy as a result of the influence of missionaries. In some tribes a woman is deserted by her husband if she becomes pregnant, because there is no other wife to carry out her domestic and agricultural duties. For the same reason a woman sometimes continues to work until she is in an advanced stage of pregnancy. Abortion is frequent from this cause and no premature infant survives among native people. In regard to infanticide, Melanesians have for generations terminated pregnancy by the use of many plants, most of which owe any potency they possess to the presence of tannic acid, or by mechanical means such as the prolonged pounding of the pregnant uterus through the abdominal wall. It was found that eighteen mothers died in every thousand births; in some areas this figure was as high as 65‰. Even when children are successfully born, they have to face many hazards, such as faulty feeding, early weaning, neglect and malaria. The training of native nurses and midwives has usually been attended with little success, owing partly to the type of woman undertaking the work and partly to the extremely low status of woman in most Melanesian communities. The best results are expected from activities instituted by missions to which the native looks with a confidence not associated with his viewpoint in other directions. A description is given of the work of committees of native women formed in the villages under the protection of the chiefs and at the urging of the Government. This consists of a kind of public infant welfare clinic. It is founded upon a knowledge of the psychology of the native and is described as already becoming extremely valuable. The activities for the care of pregnant women, for the feeding of infants and for the safeguarding of children up to the age of puberty represent the most direct attack upon the problem of depopulation observed by the mission.

It is concluded in general that a thorough study of the causes of depopulation with correction of such as can be corrected, the active institution of measures for the preservation of infant life by antenatal education of mothers and by the reduction of infantile mortality, the rapid dissemination of medical aid by a continually expanding programme of routine medication and sanitation and the furthering of research and of native medical education will provide means for the adequate control of health in Melanesia.

British Medical Association News.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Society's Rooms, Tasmanian Museum, Hobart, on February 22, 1930, DR. G. E. CLEMONS, the President, in the chair.

Annual Report of Council.

The Honorary Secretary, DR. J. H. B. WALCH, presented the annual report of the Council.

The Branch Council has pleasure in presenting the following report of the work of the Branch for the year 1929-1930.

Membership.

The membership of the Branch which was 86 at the end of last year, is now 80. Two new members have been elected and eight have transferred into the Branch, while nine have left the State, one has resigned, and five have lapsed through non-payment of the subscription, but still have the right of renewing their membership, and one member has died.

Meetings.

The annual general meeting, three special general meetings and six ordinary general meetings have been held during the year. The average attendance at these meetings was fifteen.

Papers have been read by:

Dr. F. Short: "On Tuberculosis, Osteomyelitis and Specific Disease of the Bones and Joints from the X Ray Point of View."
Dr. E. A. Rogers: "Fractures of the Spine."
Dr. T. H. Goddard: "The Historical Aspect of Venereal Disease."

A discussion on the diagnosis of acute diseases of the bones and joints in children was opened by Dr. F. W. Fay and Dr. T. Butler. Clinical cases were discussed at other meetings.

Meetings of Branch Council and Attendance.

Twenty-two meetings of the Branch Council have been held, the attendances being as follows:

Dr. G. E. Clemons	1
Dr. F. W. Fay	21
Dr. G. Sprott	16
Dr. A. W. Shugg	15
Dr. T. Butler	15
Dr. S. Gibson	14
Dr. H. W. Sweetnam	14
Dr. A. Giblin	11
Dr. J. S. Reid	14
Dr. A. Pryde	0
Dr. J. H. B. Walch (Honorary Secretary)	22

Public Hospital

The Branch and the Branch Council have been throughout the year carrying on negotiations with the Hobart Public Hospital Board with a view to the reestablishment of an honorary system in the hospital. One difficulty after another has been dealt with and overcome and the Branch has agreed to very considerable modifications of its previous standpoint with regard to recognition of the present hospital staff. The negotiations have now progressed to the discussion of details of the proposed staff and there

is every reason to believe that the Branch may soon be invited to pass the final resolution which will permit members to accept appointments at the hospital.

Visitors.

Two notable visitors have been entertained by the Branch during the year. Sir Charles Ballance who visited Tasmania at the invitation of our Post-Graduate Committee, gave us a very interesting series of lectures and demonstrations, while Sir Ewen Maclean, the immediate Past-President of the Association, and the representative of the Council, paid an official visit to the Branch and gave the Branch Council the benefit of his advice on the hospital question and other matters.

In order to provide for these and similar visits which may occur in the future, the Branch Council has appointed a permanent Entertainment Committee which consists of the President, Vice-President, President-Elect, the Chairman of the Post-Graduate Committee, and the Honorary Secretary.

Delegates et cetera.

The following delegates and representatives have been appointed by the Branch and Branch Council during the year:

Dr. F. J. Stansfield, delegate to the annual meeting of the Association at Manchester.
Dr. G. Sprott, representative of the Branch at the Sir George Syme memorial meeting in Melbourne.
Dr. T. Butler and Dr. W. K. McIntyre, delegates to the Conference on National Insurance held in Sydney.
Dr. W. Giblin, Dr. H. W. Sweetnam and Dr. A. W. Shugg, to represent the Branch in the conferences with the Hobart Public Hospital Board.

Dissecting Room.

After considerable delays and difficulty the Branch Council has been able to place at the disposal of members a dissecting room and cadaver; but, except for a demonstration by Sir Charles Ballance, no use of these facilities has been made by members.

Election of Office-Bearers.

The President announced the result of the election of office-bearers and members of the Council for the ensuing year as follows:

President: Dr. T. C. Butler.
President-Elect: Dr. J. A. Newell.
Vice-President: Dr. H. W. Sweetnam.
Honorary Treasurer: Dr. J. S. Reid.
Honorary Secretary: Dr. J. H. B. Walch.
Members of Council: Dr. G. E. Clemons, Dr. F. W. Fay, Dr. S. Gibson, with the following *ex officio* members: Dr. G. Sprott, Dr. A. W. Shugg (Representatives to the Federal Committee), Dr. W. P. Holman (Honorary Secretary of the Northern Division).

President's Address.

DR. G. E. CLEMONS gave an address on the position of the general practitioner.

Induction of President.

Dr. G. E. Clemons then vacated the chair in favour of Dr. T. C. Butler, the incoming President.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Walker-Taylor, Philip Neville, M.B., B.S., 1927 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.
Smith, William Frederick, M.B., B.S., 1927 (Univ. Sydney), "Glenevie," Firth Street, Arncliffe.

THE undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Finn, Hubert Clement, M.B., Ch.M., 1926 (Univ. Sydney), Canowindra, New South Wales.
Gillies, Gladstone Russell, M.B., Ch.M., 1924 (Univ. Sydney), 3, Bondi Road, Waverley.
McNamara, Matthew Joseph, M.B., B.S., 1928 (Univ. Melbourne), 30, Park Avenue, Mosman.
Pye, Walter Osmond, M.B., B.S., 1928 (Univ. Sydney), 43, Wigram Street, Harris Park.
Spark, Torry Ernest Hester, M.B., B.S., 1926 (Univ. Sydney), Gordon Road, Gordon.
Walker, Milner Frederick Elford, M.B., B.S., 1927 (Univ. Sydney), 12, Karori Flats, Elizabeth Bay.
Walker-Taylor, Philip Neville, M.B., B.S., 1927 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the University of Adelaide on April 4, 1930.

Islands of Langerhans.

DR. RAY HONE said that the fact that there were two distinct groups of cells in the islets of Langerhans had been well known for a long time, but owing to the special methods required to demonstrate them it was not often that a chance of actually seeing them arose.

He showed a specimen from a pancreas of a guinea-pig. The pancreas had been put into Bensley's special fixative within a few minutes of the death of the animal and the sections had afterwards been stained with Jenner's stain. The two types of cells were easily distinguishable by the different staining reactions, even under a low magnification. Dr. Hone said that it was necessary to obtain the pancreas within a few minutes of death or, owing to self-digestion, the granules and differential staining were lost.

This was shown by the next specimen from a pancreas obtained a couple of hours after death in which no differentiation could be made out. It was also observed that the cells had undergone autolysis.

The third specimen shown was obtained from a patient who had died of tuberculous meningitis. In this case the material had been obtained within ten minutes of death and put through the same process as in the case of the guinea-pig's pancreas. It also showed the two types of cells.

Two other specimens were shown; one was a section from the pancreas of a diabetic obtained at *post mortem* examination in the ordinary fashion some hours after death and fixed and stained by the ordinary methods. It was difficult to find the islets, much less to note the different types of cells in them or any pathological changes in them.

The last specimen was from the pancreas of a diabetic patient obtained within a few moments of death and fixed and stained by the special methods. Dr. Hone said that in his opinion a careful examination would disclose the degeneration of the β cells of the islets with in places vacuolation.

Post-Graduate Work.

LECTURES IN MELBOURNE.

As already announced, the Melbourne Permanent Committee for Post-Graduate Work has arranged with Professor Evarts Graham, Professor of Surgery, Washington University School of Medicine, St. Louis, United States of America, to deliver a course of six lectures in Melbourne in July next.

Professor Graham's work has attained a world-wide reputation and has covered a very large field. His researches into the physiology and pathology of the liver and gall bladder have been of special importance and have led directly to the application of the method of cholecystography to the diagnosis of diseases of the biliary tract. More recently his surgical work in connexion with diseases of the chest have contributed largely to the advancement of the surgery of that region.

The syllabus of lectures is as follows:

1. Thursday, July 17.—"The Significance of Changed Intrapleural Pressures to the Problems of Empyema and Other Thoracic Diseases."
2. Friday, July 18.—"Pulmonary Suppuration."
3. Monday, July 21.—"Surgical Treatment of Pulmonary Tuberculosis."
4. Tuesday, July 22.—"Physiology and Pathology of the Gall Bladder."
5. Thursday, July 24.—"Cholecystography."
6. Friday, July 25.—"The Clinical Significance of Tests of Hepatic Excretion."

These lectures are quite distinct from the annual refresher course. The fee for attendance at the lectures will be four guineas.

ANNUAL REFRESHER COURSE IN MELBOURNE.

THE annual refresher post-graduate course arranged by the Melbourne Permanent Committee for Post-Graduate Work will be held concurrently with the course of lectures to be delivered by Professor Graham. It will occupy two weeks, commencing on Monday, July 14, and finishing on Saturday, July 26, 1930. The fee for this refresher course will be three guineas.

Arrangements have been made with the Committees of Management of the Melbourne and Alfred Hospitals whereby a limited amount of accommodation will be available at these hospitals for those attending the course at an additional fee of three guineas per week to cover board and lodging.

It is requested that those proposing to attend these courses and those who wish to take advantage of the hospital accommodation offered, are asked to send their names and fee as soon as possible to the Honorary Secretary, Melbourne Permanent Committee for Post-Graduate Work, 12, Collins Street, Melbourne, C.1.

Correspondence.

HIGH FREQUENCY OSCILLATIONS.

SIR: As regards Dr. Charles J. Sabergerg's valuable suggestion of the use of high frequency oscillations in malignant disease I would like to mention that I have been using them for over twenty years as an adjunct to the X rays and have found them of considerable assistance. My method being after using the X rays to a safe limit, I finished the case with high frequency *effleuve*. This seemed to prolong the effect of the X rays, especially in rodent ulcer where it is quite possible to overdo X ray applications, and high frequency I found could be applied for some time with perfect safety. High frequency has fallen out of use sadly after quite a boom from the makers, principally, I think, because a nice-looking small apparatus was put on the market which was no better than a toy, and I believe hundreds were sold and except for their suggestive value of no therapeutic benefit; they were supplied with vacuum electrodes which fluorescing blue gave rise to the delusion that they were in some way similar to ultra-violet rays. The current produced was small and quite harmless, which was perhaps their chief charm. Besides its use in the above cases I have found the high frequency of some value in other conditions, particularly lumbago, sciatica, neuritis and many nervous conditions. In these cases a fairly large current must be used to have any effect. The pattern of high frequency apparatus usually sold is not the most suitable; it either gives too long a spark which is painful, or, as aforesaid, no spark worth mentioning. Mine gives about a half-inch spark and the *effleuve* is given off just a little beyond that. Also a condenser can be applied or a metallic electrode of suitable shape. When a considerable current can be borne without burning or pain, the effect is similar then to diathermy, but the difference is due to the oscillations being more rapid and voltage greater. Only a sensation of warmth is felt by the patient which seems to have a very soothing effect in painful conditions. Very small growths, such as warts, can easily be removed by the high frequency spark in a single application.

Yours, etc.,

T. G. BECKETT.

Melbourne.
April 22, 1930.

DIATHERMY OF TONSILS.

SIR: Dr. Kent Hughes's letter of May 10 *re* diathermy of tonsils might lead one to suspect that I had barely been keeping within the bounds of truth. If he had read my letter in April 12 issue carefully, there would have been no reason for any comment. No mention was made of an "Oudin" current being mentioned. With the current I employ off the Schall machine I am able to enucleate a tonsil within three minutes and the patient go away smiling, although I prefer to do it in two stages, as I think the general discomfort is less. However, I was able to show Dr. Kent Hughes that a good lump of tough beefsteak could be cut in two in a minute or so by the unipolar method. Under such circumstances one could hardly be blamed for quoting "There are more things in heaven and earth, Horatio, than are dreamt of in your philosophy."

Yours, etc.,

SYDNEY PERN.

12, Collins Street,
Melbourne.
May 15, 1930.

Obituary.

FRANCIS WILLIAM WATSON MORTON.

DR. FRANCIS WILLIAM WATSON MORTON whose death on January 26, 1930, was recorded in a previous issue, began his medical studies at the University of Melbourne and later went to Edinburgh where he obtained the diplomas of licentiate of the Royal College of Physicians and of Surgeons in 1880.

He was registered by the Medical Board in Victoria on November 7, 1884, and conducted a practice in Fitzroy for many years. He held appointments as Honorary Surgeon to Out-Patients and later to In-Patients at the Women's Hospital, Carlton.

At the Women's Hospital and as Health Officer to the City of Fitzroy for many years he was known for his enthusiasm and sterling work. Apart from his medical work and his municipal duties his chief interest was in sport. He was a keen footballer in his younger days and played tennis until shortly before his death. Valued and respected by his many friends and trusted by his patients, his loss will be widely felt and we extend our sincere sympathy to his widow and to the son and daughters who survive him.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

George, Noel Francis, M.B., B.S., 1928 (Univ. Sydney), Brisbane.
Sippe, Clive Henry, M.B., Ch.M., 1926 (Univ. Sydney), M.R.C.P., 1929 (Univ. London), Brisbane.
Beale, John George Morris, M.B., 1916, Ch.M., 1918 (Univ. Sydney), Toowoomba.
Sherman, John Burrowes, M.B., B.S., 1927 (Univ. Melbourne), Gordonvale.

Restorations to the Register:

Patrick, James King, M.B., Ch.B., 1901, B.Sc. (Public Health), 1911, M.D., 1913 (Univ. Glasgow), Brisbane.
Bonney, Henry Claude, M.B., Ch.B., 1925 (Univ. Aberdeen), Brisbane.

Books Received.

THE SOYA BEAN AND THE NEW SOYA FLOUR, by C. J. Ferrèe; 1929. London: William Heinemann (Medical Books) Limited. Foolscap 4to., pp. 90, with illustrations. Price: 6s. net.

HYGIENE FOR NURSES, by John Guy, M.D., D.P.H., F.R.F.P. & S., F.R.C.P., and G. J. I. Linklater, O.B.E., M.D., D.P.H., D.T.M. & H., M.R.C.P.; 1930. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 218. Price: 5s. net.

DIE FUNKTION DER WEIBLICHEN GESCHLECHTSORGANE UND IHRE BEZIEHUNGEN ZUM GESAMTORGANISMUS FÜR ÄRZTE UND STUDIERENDE, by Alexander V. Fekete; 1930. Berlin: S. Karger. Crown 4to., pp. 285. Price: Mk. 10.70 net.

RADIUM AND CANCER (CURIETHERAPY), by Duncan C. L. Fitzwilliams, C.M.G., M.D., Ch.M., F.R.C.S.; 1930. London: H. K. Lewis and Company Limited. Demy 8vo., pp. 180, with eight plates (four coloured) and sixty-four illustrations in the text. Price: 12s. 6d. net.

Diary for the Month.

JUNE 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 3.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
JUNE 4.—Victorian Branch, B.M.A.: Branch.
JUNE 5.—South Australian Branch, B.M.A.: Council.
JUNE 10.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 12.—Victorian Branch, B.M.A.: Council.
JUNE 12.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 13.—Queensland Branch, B.M.A.: Council.
JUNE 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 18.—New South Wales Branch, B.M.A.: Section of Obstetrics and Gynæcology.
JUNE 18.—Queensland Branch, B.M.A.: Eye, Ear, Nose and Throat Section.

Medical Appointments.

Dr. W. H. N. Randall (B.M.A.) has been appointed Government Medical Officer to the Jubilee Sanatorium at Dalby, Queensland.

* * *

Dr. Walter Lockhart Gibson (B.M.A.) has been appointed Honorary Ophthalmic Surgeon to the Brisbane Hospital, Brisbane, Queensland.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

CITY OF BENDIGO, VICTORIA: Medical Officer of Health.
COMMONWEALTH DEPARTMENT OF HEALTH: Medical Officer.
JANEFIELD SANATORIUM, BUNDOORA, VICTORIA: Resident Medical Officer.
MELBOURNE HOSPITAL, VICTORIA: Honorary Vacancies.
RENWICK HOSPITAL FOR INFANTS, SUMMER HILL, NEW SOUTH WALES: Honorary Physician.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer, Temporary Honorary Assistant Anæsthetist and Relieving Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members accepting apopintments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing. Brisbane United Friendly Society Institute. Mount Isa Hospital.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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